



Manchurian Plague Prevention Service  
Reports, 1923–1924.

〈Being Volume IV of the Series〉

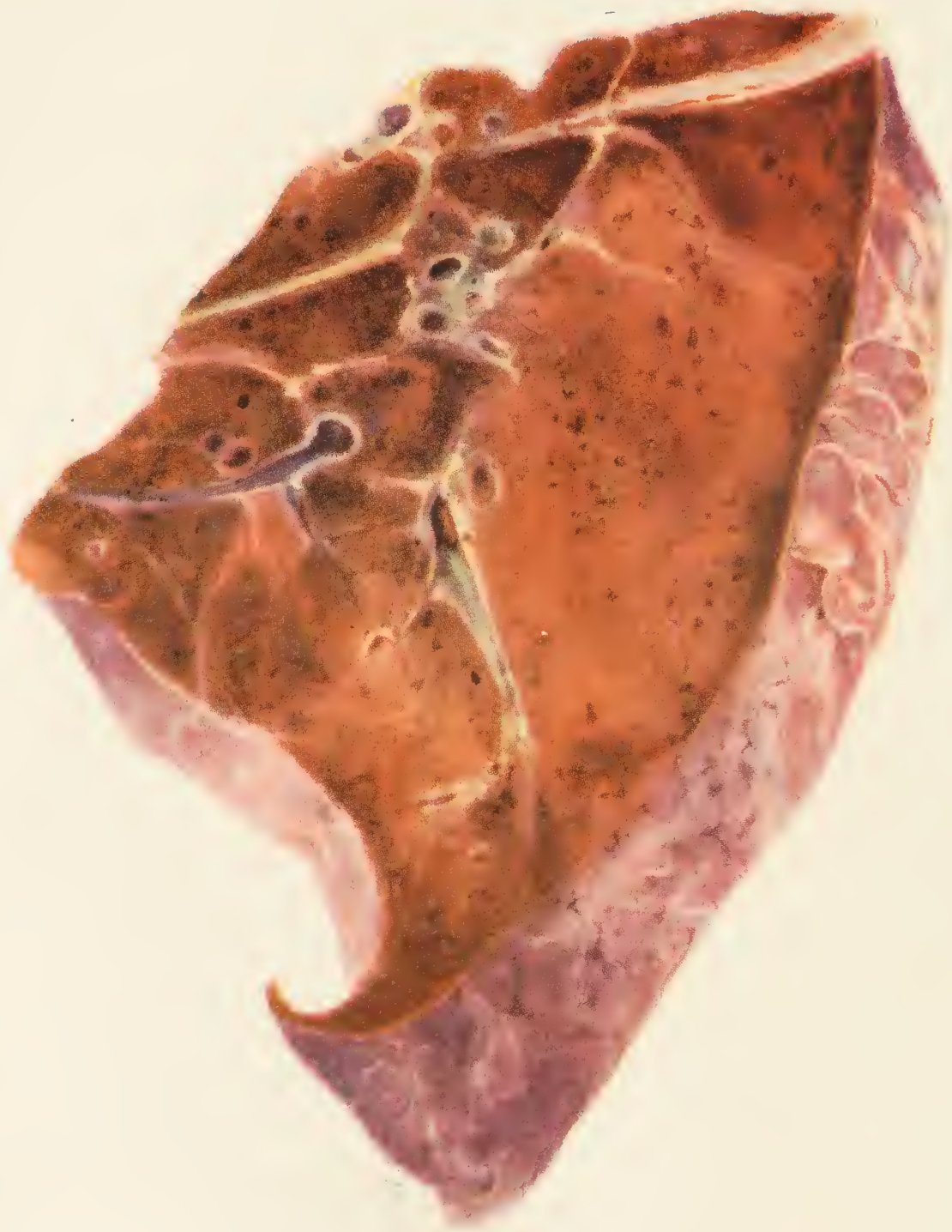


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### LUNG IN PNEUMONIC PLAGUE (Natural size and colour)

Left lower lobe cut across to show hepatized areas (right) and early infiltration (left). Fibrinous exudate over the pleural surface is well shown.

肺臟內肺疫之一部 實物大及色

- (a) 左下葉斷面之表示 (b) 肺硬變部(右) 早期浸潤(左)  
(c) 纖維性滲出物在肺胞面甚著明

# NORTH MANCHURIAN PLAGUE PREVENTION SERVICE REPORTS

〈1923–1924〉

〈Being Volume IV of the Series〉

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Grant Periodical (Curators' Rev)





科學輸入垂五十年中國能以學  
者資格與世界相見者伍星聯博士  
一人而已往者有日俄爭長於遼瀋乘疫  
事起思撰表章權政府乃置防疫公  
所於哈爾濱而以星聯總其事既逾  
十稔諸廢具舉學術上之設施六表  
以駢進於是知星聯非特優於學乃  
其治事亦不啻大過人者也星聯與



予子婿周希哲夙相知多因之以納交  
十年以來歲恒一二見：輒相與論學論  
事英述於心也星聯不自滿假又將教  
筵西邁求新知行有日矣值以所不四次  
報告冊成七序於余余不知醫術何能贊一  
言謹略述星聯之學及其信之期尚  
賡行云尔

民國十三年五月新會梁石如



# PREFACE

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In bringing out the Fourth Series of the Reports of the North Manchurian Plague Prevention Service, I wish to express my deep appreciation of the loyal cooperation and unstinted efforts of my staff who have made possible a publication of this nature after an interval of less than two years since the last Reports (1918-22) appeared.

To those who understand the difficulties under which scientific workers have to carry out their task in this country, the appearance of this Volume, like the previous ones, will be particularly welcome. In response to several requests, I have written and included in this Report an article on 'How I built Hospitals in China' mentioning therein the main incidents connected with these undertakings.

In passing, I may add that throughout the last sixteen years during which I have served the Government, I have emphasised the need of faith, perseverance and originality: the first, because without it during these troublous times one would simply have to throw up one's hands and despair; the second, because no great scientific or medical benefit has yet been achieved without a thorough attention to accuracy and details; the third, because with a conservative education handed down for four thousand years like ours, it is most essential for our minds to branch out in new directions so as to cope successfully with the progressive tendencies of the times. In spite of some faults in her ancient civilisation and the insatiable money-grabbing proclivities of many present-day leaders, the heart of China is still sound. Though apparently quarrelsome, the country is on the whole stable, and awaits only the right helping hand to put her straight. She needs true friends from outside as well as inside, and her

well-wishers (official and non-official) should encourage her best sons and daughters in their uphill work of reconstruction. Among these, educationists and scientists may be classed.

Hence, I have deemed it a great privilege in obtaining the cooperation of Professor Akira Fujinami, the eminent Head of the Pathological Institute at the Imperial University of Kyoto, in the article on 'A Study of the Morbid Histology of the 1921 Manchurian Plague Epidemic.'

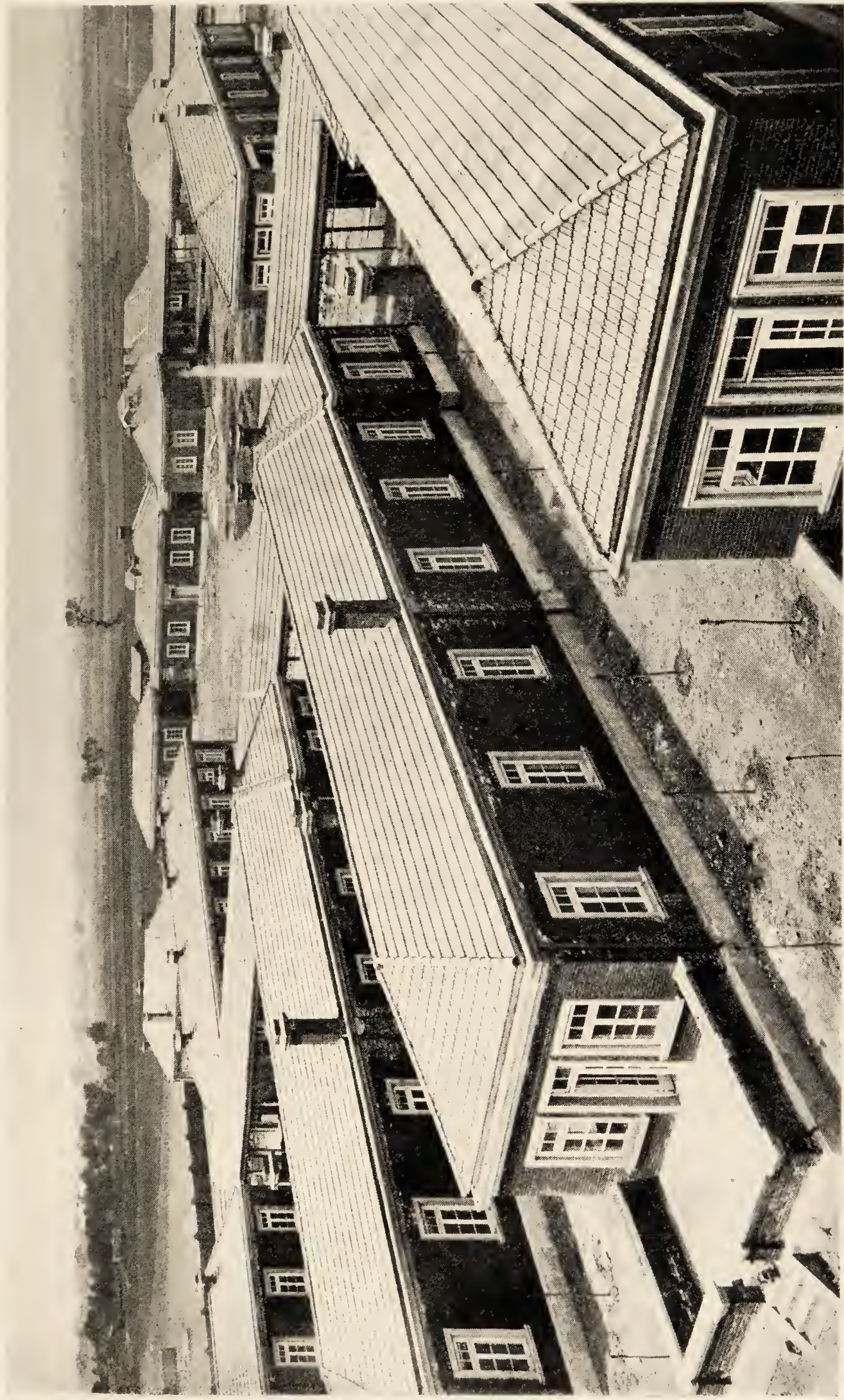
The Honorable Liang Chi Chiao, best known of China's living scholars and once Minister of Finance, has kindly written a Foreword for the Chinese edition of this Report. His composition is so fine and his caligraphy so distinguished, that I have inserted a reproduction of the original script in the English edition.

As in the Reports of 1918-22, I wish to mention specially the names of Drs. Chun Wing Han and Robert Pollitzer for their zeal and devotion. Dr. Lin Chia Swee has ably performed the task of editing the Chinese edition, which is published by the Kuang Hua Press. The coloured plates have been done by the Commercial Press of Shanghai. As usual, the Tientsin Press, Ltd., have accomplished their job in a praiseworthy manner.

WU LIEN-TEH.

Harbin, June 10th, 1924.





Birds-Eye View of New North-Eastern Hospital (in part), Mukden, completed 1924.  
Cost \$600,000.

(落成年三十民) 圖覽一望遠院醫北東天奉







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**A RECORD OF PNEUMONIC PLAGUE OUTBREAKS  
THROUGHOUT THE WORLD FROM THE  
EARLIEST TIMES.**

(WITH ONE MAP.)

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**CONTENTS.**

- I. INTRODUCTION.
- II. PNEUMONIC PLAGUE UP TO THE BEGINNING OF THE 19TH CENTURY.
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## I. INTRODUCTION.

In the course of our investigations during the last ten years or more we have often been hampered by our lack of knowledge in regard to events connected with epidemics of pneumonic plague in other parts of the world. Almost all publications concentrate upon events in the localities of the workers, and a general literature available to all has not been possible.

Hence we have attempted to collect material from every available source dealing with this subject. The result of our labours is embodied in this article. We are particularly glad to be able to submit a summary of the valuable work done by Russian investigators which up to now has been almost a sealed book to English readers.

Though far from complete, it is our hope that this article may serve as a guide to those working in the same field and may be augmented from time to time by other investigators dealing with the same problem in other parts of the world.

This compilation would not have been possible without the kind and ungrudging assistance of numerous fellow-workers. In this connection we wish to mention specially the names of Dr. G. W. McCoy (Surgeon General of U. S. Public Health Laboratory, Washington), Lt.-Col. S. R. Christophers



(Head of the Kasauli Institute, India), Lt.-Col. F. H. G. Hutchinson (Public Health Commissioner, India), Dr. R. Maresch (Prof. of Morbid Anatomy, Vienna), Dr. L. Otten (Head of the Plague Institute, Java) and the Director General, Department of Public Health, Egypt, besides colleagues of all nationalities in China, Manchuria and Siberia. To one and all we tender our best thanks.

## II. PNEUMONIC PLAGUE UP TO THE BEGINNING OF THE 19TH CENTURY.

### 1. PERIOD BEFORE THE BLACK DEATH.

In the earliest records of plague nothing is mentioned which would suggest the presence of pneumonic features. The outbreaks generally considered as authentic were apparently all of the bubonic type; this is as true of the two outbreaks described in the Bible<sup>(1, 2)</sup> as of those mentioned by Rufus of Ephesus.<sup>(3)</sup>

Some authors are inclined to classify other epidemics on record as pneumonic plague. Batzaroff<sup>(4)</sup> who is not very critical in this respect mentions:—(a) the plague during the reign of Marcus Aurelius (A. D. 164-180) described by Galen and (b) the epidemic alluded to by St. Cyprian, North Africa, in his *sermo de mortalitate*. The Athenian outbreak, 430 B.C., recorded in prose by Thucydides<sup>(5)</sup> and in poetical form by Lucretius<sup>(6)</sup> was also placed in this category.<sup>(7)</sup> It is, however, more than doubtful if these epidemics were plague at all. Hippocrates<sup>(8)</sup> mentioned lung symptoms often occurring in bubonic fevers, but we are not quite sure if he referred to true plague.

The first reference to real pneumonic plague in our opinion is that known as Justinian's plague, starting about A. D. 542 and lasting 52 years. Procopius<sup>(9)</sup> states that in Constantinople "many dropped down from a sudden vomiting of blood."

(1) 1 Samuel, ch. V & VI.

(2) 2 Kings, ch. XIX & XX; 2 Chronicles XXXII; Isaiah, ch. XXVII & XXVIII.

(3) Oeuvres de Oribase, ed. Bussemaker & Daremberg, Paris 1851, iii, p. 607.

(4) Ann. Inst. Pasteur, 1899, May.

(5) I, ii. ch. 47-54.

(6) I, VI.

(7) Vaughan, Epidemiology & Publ. H., 1923, p. 771.

(8) Sticker, Die Pest, Giessen 1908, Vol. I, p. 19-20.

(9) De Bello Persico, I, ii, ch. 22-23.

This sounds suggestive and an authority like Crawford<sup>(10)</sup> is inclined to think that those sufferers might have been attacked by pneumonic plague. It is true that Procopius emphasized that "neither the physician nor layman caught the disease by touching the sick, for many who attended upon or buried others, contrary to general expectation remained unharmed at their post. . . ." If we were to take this statement at its face value, it would certainly speak against a high prevalence of pneumonic plague at the time. It is rather questionable how far Procopius can be trusted in this respect. He was severely criticised by Gibbon<sup>(11)</sup> who quoted Evagrius' evidence against him; Crawford remarks very aptly that "the belief of Procopius in the pestilence as a special act of God almost necessitated his being a non-contagionist." It is quite probable that other foci of this pandemic, of which no records are extant, also displayed pneumonic features; contemporary records state that the disease raged at all seasons of the year and visited different places with varying fatality ("it raged so vehemently in some cities that all the inhabitants thereof were dispatched: with other towns it dealt most gently and mildly" Evagrius<sup>(12)</sup>). This high virulence and mortality might well point to pneumonic plague.

It seems to us that it would be safe when dealing with epidemics in those remote periods, to assume the critical attitude of an ancient writer, Josephus<sup>(13)</sup> who remarks: "Nor did they die after one and the same manner nor so that it was easy to know what the distemper was."

We can find no other records of pneumonic plague up to the time of the Black Death with the exception of one epidemic in Provence (A. D. 1329), where the victims coughed up blood and died on the fourth day (Cayla).<sup>(14)</sup>

## 2. THE BLACK DEATH.

The features of this unique pandemic seemed so different from those of the epidemics recorded before or since that for a long time it was not classified as plague at all.

The first modern author who maintained the Black Death to be true plague was Hecker.<sup>(15)</sup> This was fully confirmed through the researches of Webb,<sup>(16)</sup> who emphasized that symp-

(10) Plague and Pestilence in Literature & Art, 1914, p. 81.

(11) History, vol. iv, ch. XIII.

(11) History, vol. iv, ch. XLIII.

(12) lib. IV, ch. XXVIII.

(13) Antiquities of the Jews, book VI, ch. i.

(14) Qu. by Sticker, l. c., p. 41.

(15) Der schwarze Tod im 14. Jahrh., Berlin, 1832.

(16) Pathologia Indica, London, 1848.



toms similar to those of the Black Death had been noted in India during the plague outbreaks in the first half of the 19th century. Their evidence was clear, yet it was but slowly accepted for two reasons:

- (a) that the origin of the pandemic seemed different from that of other plague epidemics.
- (b) that its symptoms seemed quite atypical of plague.

(a) *Origin of the Black Death.*

A survey of this interesting problem will be found in another paper contained in this volume.<sup>(17)</sup> It may be added here that the apparent origin of the pandemic, i.e., Inner Asia, which so puzzled former authors, seems nowadays a strong argument in favour of its true nature.

(b) *Unusual symptoms.*

It is undeniable that some of the symptoms of the Black Death are not frequently encountered in modern outbreaks of plague, while others are altogether absent.

Under the first group come the skin manifestations, described by Boccaccio<sup>(18)</sup> as follows: "It was the quality of the disease to show itself by black or blue spots, which would appear on the arms of many, others on their thighs, and every part else of the body—in some great and few; in others, small and thick." Jennings<sup>(19)</sup> analysing the old records, comes to the conclusion that these signs are "really identical with the blebs or blisters so commonly observed in recent epidemics." This possibility has to be admitted, but we must point out in addition that larger and smaller skin haemorrhages were noted in some bacteriologically confirmed plague outbreaks.

Our difficulties are greater when we endeavour to analyse the other group of symptoms peculiar to the Black Death, such as, gangrenous inflammation of the lungs with offensive breath, as described by Simon de Covino<sup>(20)</sup>, who fought the disease at Montpellier and by Cantacuzene who witnessed it at Constantinople. Mueller,<sup>(21)</sup> who made a careful study of pneumonic plague to which he himself succumbed, is inclined to think that this was a real symptom and not the product of the excited imagination of contemporary observers. He states that occasionally commencing necrosis of the lungs

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(17) The Original Home of Plague. Historical Evidence.

(18) The Decameron.

(19) A Manual on Plague, London, 1903, p. 8.

(20) Qu. by Haeser, Gesch. d. Medizin, Jena 1882.

(21) Die Pest, Wien 1900, p. 225.

could be noted in modern post mortems and quotes as examples two cases of Sticker. The majority of modern authors, including Mueller himself, agree that they could not perceive any characteristic smell in plague sufferers, although much was made of that sign by old recorders. On the other hand, it is possible that the Black Death was due to a mixed infection: the two instances mentioned by Mueller were cases in point. Kolle<sup>(22)</sup> states also "that some observers wish to ascribe the appearance of larger carbuncles in plague to a mixed infection."

In a recent contribution<sup>(23)</sup> we found the interesting hypothesis that the atypical features of the Black Death might be due to co-existing scurvy. The fact that the Black Death does not quite correspond to the form of plague as it is known to-day cannot eliminate the ample evidence, that it was plague. The descriptions of both the bubonic and the pneumonic types, as given by the contemporary observers, leave no room for doubt. We shall not deal with the bubonic form, but it is perhaps necessary to emphasize the large part this type played in the Black Death because it is occasionally mentioned as being of purely pneumonic character.

It can be gathered from the records of contemporary writers that the pneumonic type was most prevalent at the beginning of many local outbreaks, that only in some localities it continued to rage in this form, but that most often it assumed a bubonic character after a few months. Sticker<sup>(24)</sup> is inclined to think that this early preponderance of pneumonia was observed whenever the outbreaks started in winter and that the change to the bubonic form occurred in spring. This rule, however, did not always hold; thus we read in the Pskov chronicle<sup>(25)</sup> of a big and purely pneumonic outbreak in 1352: "it began in spring, in the blossom week, lasted to the very fall, ended before winter."

Recorders of the 14th century differentiated clearly between pneumonic and bubonic plague as manifestations of one and the same disease and also noticed that those with lung affections died quicker than the others. Several authors state that all with bloody sputum died; an exception appears in a statement by Emperor Cantacuzenus, that a few exhibiting all features of the disease recovered contrary to expectation. Those who recovered were said to have "all the features of the disease," hence the pneumonia present was probably secondary to bubonic infection.

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(22) Wilson, Infect. Dis., 1911, p. 747.

(23) Egyptian Pl. Rep., 1923, p. 64.

(24) L. c. p. 73.

(25) Florinski, Collection of Plague Papers, Kasan, 1879.



Guy de Chauliac<sup>(26)</sup> also emphasizes the greater infectivity of the pneumonic type.

A few instances of outbreaks during the Black Death may now be given. These are necessarily confined to Europe, as no satisfactory records about the East can be obtained.

The presence of pulmonary symptoms in the Constantinople invasion has been referred to.

*Italy*:—Boccaccio speaks only of bubonic and skin symptoms in Florence. In many places bloody expectoration was noted as well, such as Venice (winter and spring 1347)<sup>(27)</sup>, Istria and Friaul (August 1348),<sup>(27)</sup> Noricum Cisalpinum (1348),<sup>(28)</sup> Piacenza (from summer till end of 1348)<sup>(29)</sup> and Sicily (autumn 1347-April 1348).<sup>(30)</sup>

*France*:—Allusion has been made to Guy de Chauliac's classic description of the pest in Avignon (Jan. 1348). His clinical observations were supplemented by *post mortems* in proving affection in the lungs.

*Spain*:—The presence of the pneumonic type is testified by Arabian writers (Ibnul Khatib, etc.)<sup>(31)</sup>.

*British Isles*:—The first outbreak in England "was evidently one of what is now called pneumonic plague."<sup>(32)</sup> Greenwood<sup>(33)</sup> believes that the heavy mortality at the beginning of the London epidemic was caused by the pneumonic type. Friar Clyn's Annals of Ireland<sup>(34)</sup> show that Ireland did not escape this worst of all scourges.

*Poland*:—Plague was introduced there at the end of January 1349 from Hungary; it raged in pneumonic form till March and continued up to the autumn as bubonic plague.<sup>(35)</sup>

*Russia*:—The pneumonic form seems to have been more predominant and persistent.<sup>(36)</sup> But here also the bubonic type played a role; the Nikolaevsk chronicler for instance relates that "the people coughed up blood and others showed affected glands."<sup>(37)</sup>

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(26) La grande Chirurgie, composée en l'an 1363.

(27) Muratori, qu. by Sticker, p. 50.

(28) Dionysius Sec. Colle, *ibid.*, p. 51.

(29) *Ibid.*, p. 52.

(30) Micaelus Platiensis, *ibid.*, pp. 47-49.

(31) *Ibid.*, pp. 64-66.

(32) Payne, in Allbutt & Rolleston, A System of Med., vol. II, p. 359.

(33) Jl. Hyg., Pl. Suppl. I, p. 93.

(34) Edit. Butler, Dublin, 1849.

(35) Philippe, Histoire de la peste noire, Paris, 1853.

(36) Sticker, l. c., p. 72.

(37) Coll. works of Russian Chron., XI, p. 3.

It may be expected *a priori* that during this virulent pandemic with its large proportion of pneumonic cases those caring for the sick suffered greatly. And indeed considerable evidence is available in this respect. Thus Venice and Montpellier lost almost all the doctors during the outbreaks.<sup>(38)</sup> Chauliac's observations (see above) are confirmed by another eye witness in Avignon, complaining that "there is no known means of protection."<sup>(39)</sup> Gabriel de Mussis<sup>(40)</sup> sighs for the priests and doctors who "quickly followed the deceased." In England "those who touched the dead, or even the sick, were incontinently infected that they died" (Friar Clyn). No wonder that in some instances the sick remained unattended and the dead unburied; but on this dark background shine many examples of faithful devotion. Thus in Paris there were always new volunteers to replace the Sisters of Charity dying at their post. Morley remarks touchingly in his introduction to Boccaccio: "The age had its unnamed heroes whose fame spread only in Heaven."

Our records of pneumonic plague would not be complete without some reference to the epizootics which led to them. It is not easy to do so in the case of the Black Death, because the statements are somewhat contradictory. It appears that the continent of Europe had been infested for ages with mice and that *Mus rattus* had been introduced since the ships of the crusaders returned home.<sup>(41)</sup> Indeed we find at least two references dealing with the role played by these rodents:

- (a) Nicephorus Gregoras<sup>(42)</sup> records that during the outbreak at Constantinople besides the domestic animals "even the mice that lived within the walls of their houses" were attacked. Abel,<sup>(43)</sup> who always tries to discredit evidence in favour of rodent infection, suggests that Gregoras merely copied an old statement of Avicenna (A.D. 908-1037) of Mesopotamia.
- (b) According to Gillis di Muisis,<sup>(44)</sup> the Black Death in Flanders in summer 1349 was preceded by a fatal disease among mice, rats and dogs.

Such records as the above deserve attention though they lose much value by the inclusion of domestic animals. For some parts of Europe, however, the evidence of rodent

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(38) Mueller, l. c., p. 115.

(39) Jennings, l. c., p. 11.

(40) Qu. fr. Payne, St. Thomas's Hosp. Rep., vol. XVII, p. 11.

(41) Hinton, Rats & Mice as Enemies of Mankind, London, 1918.

(42) Hist. Byzant., lib. XVI, ch. 1.

(43) Z. f. Hyg., 1901.

(44) Mueller, l. c., p. 90.



influence is negative. Hankin<sup>(45)</sup> states that there were no rats in England at the time of the Black Death. Greenwood remarks: "that the intralocal spread of human plague is due to an epizootic cannot be seriously questioned so far as India is concerned. Nevertheless we are not warranted in asserting that this method of spread has prevailed in all epidemics elsewhere, even when the type was predominantly bubonic. For instance, in many parts of Europe, probably in English rural districts, the Black Death was chiefly bubonic plague. But I can find no trustworthy evidence that a rat epizootic was a factor in this particular pandemic." Martin's<sup>(46)</sup> statement is of great interest: "A variation of the plague bacillus in the direction of greater infectivity, with perhaps diminished toxicity leading to a higher degree of septicemia in man, would permit of direct transmission by human fleas. Bubonic plague would then be independent of the rat, and spread directly from man to man. For several reasons it seems not improbable that this may have happened in the plagues of the Middle Ages."

### 3. NOTE ON PNEUMONIC PLAGUE IN THE FOLLOWING CENTURIES.

We have stated already that pneumonic plague plays its most important role at the beginning of the Black Death. As far as we can see this seems to have subsided quickly and even permanently in some affected localities. In England the *first* epidemic was pneumonic, but the so-called second and third plagues of Edward III.—not designated as Black Death by some historians—were of another character. To quote another example, it appears that the second outbreak at Avignon (1360) was mainly, if not solely, shown as bubonic and skin affections; during the third epidemic in 1372 many recovered.

However, no general deduction can be gathered.

Kolle<sup>(47)</sup> states that from the 17th century onwards the plague epidemics were less fatal, because they were more often of the bubonic type. Mueller<sup>(48)</sup> remarks that in the first half of the 19th century the clinical features of the Black Death became so utterly forgotten that only after long literary studies was its plague nature established. There was a period when plague was only recognised in its bubonic form and undoubtedly the pneumonic type was absent. A careful study

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(45) Ann. Inst. Pasteur, 1898, pp. 746-762.

(46) Br. M. Jl., Nov. 11, 1911.

(47) Kolle-Hetsch, Exp. Bakt. & Inf.-Kr., 1919, p. 419

(48) L. c. p. 244.

of this question has been made by Mueller. The absence of the pneumonic type at that time is the more remarkable, as earlier outbreaks occurring between the Black Death and this period displayed lung symptoms, as shown by the following tabulation :

<i>Year :</i>	<i>Locality :</i>	<i>Type of Plague :</i>
1417-19	Russia (Pleskov, etc.)	Pn. and bub.
1420-21	Russia (spreading from Dorpat)	Extensive bub. and pn. Note 2.
1506	Cologne.	Pn. outbr. in winter.
1528	Elsa valley (Savoie)	Pn. outbr. in winter preceding bub.
1523	Geneva	Pn. outbr. in winter preceding bub.
1535	Venetia and Lombardy	Bub. preceded by malignant pneumonia.
1563	Gascogne and Provence	Pn. and bub. Extensive outbreak in many parts of Europe, app. introduced fr. the East. Note 1.
1564-66	Tyrol	Pn. and bub. Extensive outbreak in many parts of Europe, app. introduced fr. the East. Note 1.
1629	Basses Alpes (South France)	June to Oct. Pn. and bub.
1630	Island near Venice	Some pn. cases after bub. with secondary pn.
1635-37	North. Germany and Netherlands	All types of plague. Extensive. Note 1.
1665	London	Preceded by great incidence of respiratory diseases. During the epidemic lung changes found occasionally at p.m. Note 3.
1668	Grindelwald (Switzerland)	Bubon. and pn. cases.
1708-10	Hungary, Transylvania	Pn. prevailing during third year of epidemic.
1710	Copenhagen	Bub. and pn. Extensive.
1720	Marseilles	Secondary pneumonia frequent in bubonic.
1737-38	Otshakoff (South Russia)	Pn. and bub. Extensive.

Note 1 : At least part of these outbreaks occurred soon after plague had been re-imported into Europe from the East.

Note 2 : Derbek (49) states that both the pneumonic and the bubonic types continued to exist, often simultaneously, in Russia up to the 17th century.

Note 3 : While performing a p.m., the doctor became infected but recovered (50).

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(49) History of Plague in Russia, Breslau, 1906.

(50) Simpson, A Treatise on Plague, p. 237.



### III. PNEUMONIC PLAGUE IN INDIA, THE COAST COUNTRIES OF EAST-ASIA AND CHINA PROPER.

#### 1. PNEUMONIC PLAGUE IN INDIA.

Lung symptoms formed a distinct feature in the Indian outbreaks during the first half of the 19th century.

Gilder and Whyte, visiting Kathiawar during the 1812-21 outbreak, mentioned both bubonic and pneumonic forms in letters to the Bombay Medical Board (1820).<sup>(51)</sup> Gilder notes that the natives were well acquainted with the different aspects of pest and had a special name for each type (*Ghant no rogue* for the 'knotty' disease, *kogla no rogue* for the 'expectorating' disease; *tao no rogue* for the 'fever' disease). Whyte actually saw pneumonic cases when visiting the town Muli. He was impressed by the fact that every house was surrounded by a thorn wall higher than a man remarking "that no better means could possibly have been adopted completely to exclude ventilation, if this had been the sole object of the inhabitants." His statement that in the affected localities the pneumonic form preceded the bubonic is significant. In the 1836-38 outbreak, well known as the 'Pali plague', lung symptoms were not so much in evidence but the "pulmonary variety was sufficiently well marked to excite special attention."<sup>(52)</sup> It was noted by Forbes, by Keir and by Maclean. The Pali outbreak, caused apparently by a rat epizootic, was of a mild type till October 1837 when it became virulent in character.

Pneumonic signs were less conspicuous at Kumaon and Gurwhal. Pearson (1852) enumerates 'expectoration of blood' among other symptoms but does not mention its frequency or whether it is primary in character. There is no doubt that cases without buboes were not rare, especially at the onset of the outbreaks but, so far as ascertainable, they succumbed quickly reminding one of the septicemic type.<sup>(53)</sup> It should be remembered, however, that little was known of other than pure bubonic cases.

#### *Present pandemic.*

i. *Bombay*: Pneumonic cases were frequently seen in the early days, when Bombay was first attacked (1896.) In fact some of the first recorded cases were purely pneumonic in character.<sup>(54)</sup>

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(51) Nathan, *Plague in India*, vol. I, p. 75.

(52) *Ibid.*, p. 85.

(53) *Rep. German Pl. Comm.*, p. 15.

(54) *Rep. of Health Off. Bombay, 1896*; Zabolotny, *Pestis bubonica, 1907*.

- a) Childe investigated 12 cases from the end of 1896 to the beginning of 1897, ten of which were p.m. The other two (Dr. Manser and his nurse) were clinically observed and verified by autopsy. Dr. Manser was a busy practitioner and president of the Plague Research Committee on whose behalf he was studying the treatment by means of drugs. He fell sick suddenly with rigor on Jan. 2, 1897 and developed signs of pneumonia. Childe<sup>(55)</sup> states when referring to the case, "that the symptoms were not like ordinary pneumonia.....the patient's general condition was far worse than could be explained by the small amount of lung disease present.....So I examined the sputum under the microscope and found it full of bacilli looking like those of plague, and cultures were made from which a pure growth of the plague bacillus was obtained." The doctor died on Jan. 6. His nurse who had never attended a plague case before succumbed with similar symptoms on Jan. 10 after an illness of less than four days.
- b) The various Plague Commissions working in Bombay during 1897 recorded pneumonic cases:
- a. The Austrian Commission saw clinically 6 patients and dissected 3. Mueller, their clinician, quoting statistics from the Report on the Outbreak of Bubonic Plague in Bombay, 1896-97, gives 8% primary pneumonic cases among 939 observed in 1897 in the Arthur Road Hospital. He, however, questions the accuracy of this high pulmonary rate, and suspects the presence of hidden buboes among them.<sup>(56)</sup>
- b. The German Plague Report contains detailed reports of 6 pneumonic patients and short notes of six more.

Among the first group a chain of three cases among the lower sanitary staff of the Parel Hospital is of interest. These were three hospital cooks who succumbed to pneumonic plague at the beginning of April in quick succession. Animal experiments performed with dust from the kitchen walls, flour, etc., remained negative, hence the Commission believed infection to have been conveyed by a female plague patient whose tobacco pipe they had used and with whom they had had sexual connection.

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(55) Rep. of Surgeon-Maj. Lyons, Pres. Pl. Res. Comm.

(56) Austrian Pl. Rep., vol. I, p. 148.



The Commission notes also the death from pneumonic plague of Dr. Desai, chief-surgeon of a Hindu Hospital.

The following statement also occurs in the Report: the number of deaths from phthisis and respiratory diseases is markedly higher under the influence of the plague epidemic. Many cases of *plague pneumonia* are hidden under these diagnoses; patients with pulmonary tuberculosis are specially endangered during a plague epidemic.<sup>(57)</sup>

Other observers agreed that pneumonic plague was misdiagnosed in the early days.<sup>(58)</sup> McCabe Dallas stated that "during the early months of the epidemic the pulmonary form of the disease escaped detection and thus spread the infection unchecked."<sup>(59)</sup>

It may be added that plague deaths were indiscriminately certified under 'respiratory diseases'<sup>(60)</sup> but some of these might be classed under pneumonic plague. This same confusion exists in other countries as will be mentioned later on.

- c. Eitter of the Egyptian Plague Commission saw only one case in the European Hospital in Bombay with slight lung symptoms but no buboes; the scanty sputum showed occasionally some admixture of blood. Plague bacilli were found in the sputum on 7th day, while repeated blood tests were negative. The patient recovered after long illness. Mueller<sup>(61)</sup> is not convinced that this was *primary* plague pneumonia. Bitter believes pneumonic cases to be more frequent than is generally described and that they played an important role in the spread of the epidemic. He thinks that they may give rise not only to pneumonic but also to bubonic infection through the sputum entering the skin.

- d. The Russian Plague Commission<sup>(62)</sup>—dissecting and studying 6 pulmonary cases—also laid stress

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(57) German Pl. Rep., p. 33.

(58) Birdwood, Quart. Jl. R. Met. Soc., Jan. 1900, p. 90.

(59) Nathan, l. c., p. 17.

(60) Loc. Gov. B. Rep. 1898-01, p. 248.

(61) L. c., p. 224.

(62) Ann. Inst. Pasteur, 1897, p. 663.

upon the frequency of that type. One of their members (Zabolotny) stated in a later contribution<sup>(63)</sup> that 18-23% of the patients in the Indian plague hospitals displayed the features of pneumonic plague.

e. Simond<sup>(64)</sup> remarks upon the high incidence of pneumonic cases in 1897 and believes that they as well as all bubonic patients are infected through flea bites<sup>(65)</sup>.

c) Nathan quotes General Gatacre's statistics from the 'No. 10 district' as follows :

Enlargement of glands .....	85%
Pneumonic type .....	12%
Gastro-enteric type .....	3%

Choksy's statistics based upon 11,600 cases are :

Bubonic .....	77.65%
Septicemic .....	14.25%
Pneumonic .....	4.10%
Gangrenous skin pl. ....	2.50%
Mixed infection .....	1.00%
Pestis ambulans .....	0.50%

This percentage of 4.1% for pneumonic cases in 1896-7 is interesting as compared with 2.44% as collected by Simpson in 1902<sup>(66)</sup>. This diminution is testified by Schottelius<sup>(67)</sup> Duerck<sup>(68)</sup> and Kashkadamov<sup>(69)</sup>.

A pneumonic outbreak in Bombay 1898, described by Polverini and discussed by Di Giura at the Mukden Conference<sup>(70)</sup> deserves notice because it refers to the hospital staff and their families. This appeared at a time when the plague epidemic was declining, not more than 10 cases being noted daily. The first victim was a man engaged in the removal of dead bodies. He had undoubted pneumonic plague and succumbed in 3 days. Further developments are as follows :—

(63) *Pestis bubonica*, 1907.

(64) *Traité Path. Exot.*, Grall & Clarac, 1913, vol. VI. p. 476.

(65) *Ann. Inst. Pasteur*, 1898, p. 625.

(66) *L. c.*, p. 170.

(67) *Hyg. Rundsch.*, 1901.

(68) *D. Med. Woch.*, 1901, p. 310 (Suppl.)

(69) *Vj. Obsh. Guig.*, 1901, p. 1.

(70) *Mukden Conf. Rep.*, pp. 180-182.



		<i>Sick</i>	<i>Died</i>
2.	Nurse attending No. 1	Sept. 23	Sept. 24
3.	Hosp. Ass. „ „ 2	„ 25	„ 26
4.	His wife „ „ 3	„ 25	„ 27
5.	Dr. Choukar „ „ 2 and 3	„ 29	Oct. 2
6.	Indian Dr. „ „ 5	?	After 4 days
7.	Store „ „ 5	Oct. 5	Oct. 12
8.	Relative of No. 5	„ 6	„ 9
9.	Assistant „ „ 5	„ 9	„ 13
10.	Brother „ „ 9		
11.	„ „ „ 9		
12.	Nurse		
13.	„		
14.	„		

Total 14 cases, all fatal pneumonic.

Another instance of hospital infection occurred in a sister: A patient with lung plague coughed at her face, part of the sputum entering the conjunctival sac of one eye (Feb. 15, 1898). Although her eye was carefully washed, it smarted and felt sore until the 18th. Then she became feverish and developed an auricular bubo on the corresponding side. She succumbed on the 22nd.<sup>(71)</sup>

Zabolotny records two pneumonic outbreaks in the Bombay Presidency:

- (a) In the village Cangaur (Baroda), where from one patient 13 immediate contacts and 24 persons taking part in the funerals were attacked.
- (b) In Cutch where 10 members of a family were infected by one person<sup>(72)</sup>.

ii. *Calcutta* (seriously affected in 1899):

If the words of Hossack, an opponent of the rat-flea theory, could be believed, pneumonic plague was frequent in the early years. According to him: "In 1900 out of 194 cases observed directly by me, 32.4% showed respiratory symptoms and about 30% of the total cases investigated in my district of Calcutta did the same.....I have come to regard plague pneumonia, not as an aberrant and rare type of a disease which is local rather than generalised, with buboes marking the site of skin infection, but as a frequent and occasionally a predominant expression of a disease which is essentially a septicemia."<sup>(73)</sup> It is thus difficult to decide how far such *respiratory symptoms* corresponded to primary pneumonic plague. Hossack himself considers all types as the result of a blood infection and does not differentiate them sufficiently.

(71) Clemow, *Lancet*, May 1900, p. 1509.

(72) L. c.

(73) *Lancet*, Nov. 24, 1900; *Indian Med. Gaz.*, Aug., 1909.

In later years the number of pneumonic cases appeared few. Thus in 1910:—

Bubonic .....	529	
Pneumonic .....	28	(2.12%)
Septicemic .....	767	

Total ..... 1324<sup>(74)</sup>

In 1914 “for the first time no cases of the pneumonic type were reported<sup>(75)</sup>.

Hossack<sup>(76)</sup> stated that in the heavily affected northern quarter of the city 60-80% of the rodents were *Nesokia bengalensis* which played an important part in the spread of the infection.

iii. From Calcutta a series of outbreaks could be traced to *Eastern Bengal and Assam*. These are dealt with by the Indian Plague Commission<sup>(77)</sup>. The disease ran the following course:

Year	Season	Locality	Type No. Cases	Remarks
1898	Sept.	Backergunge	Pneum. 11 (fatal)	Introd. fr. Calcutta (78)
1899	Feb.—March	Pania villages	„ 42 (38 d.)	„
1899	March	Tippera	Bubon. 5	„
1899	March	Faridpur	Pneum. 30 (29 d.)	2 bub. c. Source?
1903	May	Dibrugarh	Bubon. 36 (22 d.)	Local rodents involv.
1906	March—April	Serajganj	Pneum. 27 (24 d.)	Introd. fr. Calcutta
1906	June	Mymensingh	Pneum. 5 (fatal)	„
1906	August	„	Pneum. 50 (45 d.)	„ (79)
1907	January	Malda distr.	Pneum. 4 (fatal)	„ fr. Ballia
1907	April	Goalundo	Bubon. 1	„ by steamer
1907	April	Tippera	Pneum. 1	„ fr. Calcutta
1907	June	Manipur	? Bubon. 8 (4 d.)	„ fr. Burmah
1909	March	Goalpara	? 1	„ fr. Calcutta
1910	Jan	Chittagong	Bubon. 1	„ fr. Unit. Prov.
1910	June	Noakhali	Pneum. 51 (45 d.)	„ fr. Calcutta
1911	?	Faridpur	Pneum. 17 (fatal)	(80)
1914	?	Goalpara	? 1	Introd. fr. Ballia (81)

It would thus seem that the outbreaks were almost all of the pneumonic type. The Indian Plague Commission points

(74) L. G. B. R. 1910-11, p. 131.

(75) Ibid., 1914-17, p. 30.

(76) Jl. Hyg., Vol. VII, Pl. Suppl., p. 703.

(77) Ibid., vol. XI. Pl. Suppl., p. 158.

(78) Simpson, p. 212.

(79) L. G. B. R. 1906-07, p. 83.

(80) Ibid., 1912-13, p. 25.

(81) Ibid., 1914-17, p. 72.



out, however, that the importation of pneumonic types was noted because they spread while bubonic cases—most probably introduced as well—attracted no attention because no epidemic followed them. This freedom from bubonic plague is apparently due to scarcity of rats in the houses.

The pneumonic epidemics developed throughout the year, but it must be remembered that “the climate of Eastern Bengal and Assam is characterised by coolness and extreme humidity.”<sup>(82)</sup>

iv. *Punjab*: Several pneumonic outbreaks were recorded in this province and—as in the case of Eastern Bengal and Assam—were introduced by human beings.<sup>(83)</sup>

Browning Smith<sup>(84)</sup> described an outbreak of pneumonic plague in one family in Jan.-Feb. 1903 at Munda Dinah brought by a man returning from the infected village of Bagrian. 15 persons succumbed in quick succession. The rat population was apparently unaffected.

The Plague Investigation Staff in Upper Egypt (1911-12) compares the incidence of the different plague types in Kous and the findings in the Punjaub villages Dhand and Kasel (1905-06)<sup>(85)</sup>:

Locality	Fatal bub. and sept.			Prim. pneum.	% prim. pn. to total c.
	without pn.	with pneum.	% with pn.		
Kous	34	8	13	12	15.2
Dhand and Kasel	49	4	4	2	2.0

Gill<sup>(86)</sup> observing four plague epidemics in the Punjab from 1905-09 came to the following conclusions regarding the pneumonic form:

“(1) Primary pneumonic plague in all its stages is intimately associated with the bubonic variety.

(2) When occurring as an original infection it is associated with a preceding rat epizootic in the same way as bubonic plague.

(82) Jl. Hyg., Vol. XI., Pl. Suppl., p. 172.

(83) James, Simpson, p. 196.

(84) Ind. Med. Gaz., June 1904.

(85) Progress Rep., Cairo, 1912, pp. 21-22; Jl. Hyg., Vol. VII. Pl. Suppl., p. 895.

(86) Ind. Med. Gaz., 1909, p. 135.

- (3) It rapidly tends to die out as such with or without being succeeded by a bubonic outbreak.
- (4) Its mode of spread is 'direct' from man to man, but owing to the readiness with which rats become infected, it is liable to give rise to a rat epizootic, which in turn gives rise to a bubonic plague epidemic.
- (5) It occurs usually and chiefly at the commencement of the epidemic season, being chiefly confined to the first three months.
- (6) That as regards individual epidemics it is mostly present at the commencement of such outbreaks.
- (7) That in estimating the effect of pneumonic plague on the general spread of the disease its power of producing bubonic outbreaks requires to be taken into consideration.
- (8) That pneumonic plague plays a definite though variable part in the specific septicemia, called plague, of which, perhaps, it forms the expression of an unusual or 'exalted' degree of virulence."

Gill adds that pulmonary plague is more common in the comparatively cool climate of Punjab than in other parts of India where warmer and damper weather prevails. In a previous paper (1908)<sup>(87)</sup> he gave some details about the 1907 outbreaks in the Thelum district. In 56 villages investigated by him, pneumonic plague prevailed in nine (16%). In all these 9 instances the infection could be traced to the arrival of one individual in the incubating stage. Recoveries from pneumonic plague occurred rarely, if ever. In 1921 "the pneumonic type was practically absent and the mildness of the epidemic was chiefly attributable to a comparatively weak monsoon, and to the preventive measures that were adopted"<sup>(88)</sup>.

v. *North-West Frontier Province*: According to Johnstone<sup>(89)</sup> 61 persons succumbed in an outbreak in the Hazara district to virulent pneumonic plague, only one doubtful case recovering.

Col. Hutchinson has kindly furnished us with notes about two later outbreaks :

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(87) Lancet, 1908, Jan. 25th.

(88) Ibid., Aug. 1922, p. 310.

(89) L. G. B. R., 1913, p. 19.



Year	Season	Locality	No. pneum. c.	Remarks
1920	March 10— April 2	Jalsal (Mar- dan tehsil)	26 (25 d.)	Introd. by pat. with sec. pn. Follow. by severe bub. outbr. in neighb. village.
1920	March 31— April 2	Jalbal, 5 m. dist. from Jalsal	17 (15 d.)	Imported through pn. c. fr. Jalsal.

vi. *Kashmir*: Mitra<sup>(90)</sup> described an extensive and mainly pneumonic outbreak in 1903-04. It arose from a bubonic case in November<sup>(91)</sup>, was very virulent from Dec. to March and lasted till August, 1904. 1443 cases were counted with 20 recoveries; the latter were of the bubonic type and occurred at the end of the epidemic. Mitra emphasizes the severity of the winter in Kashmir.

No epizootic was noted, though *M. Rattus* infested the houses<sup>(92)</sup>.

vii. *Southern Parts*: All the outbreaks recorded thus far took place in the northern districts of India, where the higher incidence of pneumonic plague was noted by Gill. These conditions have certainly not changed since his time, because Col. Hutchinson remarks to the same effect in a letter to us dated Oct. 22, 1923. He mentions also an outbreak of unknown origin with 22 cases, occurring from Feb. 15 to March 6, 1915, in Dejwar Tryli in Pomch. Stat.; we could not locate this spot on our maps.

The Indian Plague Commission states<sup>(93)</sup>: "Pneumonic plague, though highly contagious, plays an insignificant role in the spread of the disease on account of its low incidence (less than 3% of total cases)." Regarding the *Madras Presidency*, the *Tropical Dis. Bulletin* (<sup>94</sup>, 1914) says: "Both septicemic and pneumonic plague have been present in the Madras Presidency, and, in certain epidemics, this markedly has been the case and, as in Ceylon, they have *preceded* bubonic plague. Nor in that area, in well watched populations, do epizootics always precede epidemics; though the former ultimately determine the extent of the latter....."

(90) Ind. Med. Gaz., 1907, p. 133.

(91) Hossack, Jl. State Med., 1913, p. 229.

(92) Egypt. Rep. 1923, p. 44.

(93) The Etiology & Epidemiology of Plague, Calcutta 1908, p. 93.

(94) Vol. III. p. 370.

The Indian Plague Commission mentions only a few pneumonic cases as encountered there<sup>(95)</sup>. Col. Hutchinson amplifies this<sup>(96)</sup>:

Year	Season	Locality	No. total cases	No. pneum. cases	Remarks
1913	Jan. 17—20	Nilgiris (rural)	20	2	
1921	Nov. 3— March 5	Edyur Hamlet Tiruppu-vanam (Ramnad Distr.)	124	20	Introd. by bub. c. with sec. pn. from Madura.
1921	„	do	167	50	
1922	Oct. 3— Nov. 13	Ootacamund Taluk Wellington Canton.	16	9	
1922		Coonoor Town	16	2	
1922	June 15— July 22	Ootacamund Town	16	11	5 ? pneum. Introd. from Malabar.

## 2. SIAM AND INDO-CHINA.

The incidence of pneumonic plague in Siam and Indo-China is very low. Such cases occur occasionally but neither they nor the cases of tonsillar plague with free expectoration of bloody sputum teeming with plague bacilli<sup>(97)</sup> apparently infect contacts. Campbell Highet thinks this due “to the open air habits of the people and to the rapidity with which particles of mucus or blood dry in our temperature.” Similar observations were made at the Saigon Conference (1913) by Montel<sup>(98)</sup> who remarked that pneumonic cases have never created a focus. This led Manaud<sup>(99)</sup> to investigate the influence of climate upon pneumonic plague.

It may be observed, however, that the protecting influence of climate and habits of life are not quite absolute. One pneumonic outbreak at Ban Phagi, costing 22 lives, was claimed by Braddock<sup>(100)</sup> to arise ten days after a purely bubonic affection at Kerat (Siam), 200 miles away. Hostalrich<sup>(101)</sup> noted only 7 pneumonic cases among 1100 plague patients in the Bonthuan province of Annam, but five of these occurred among close contacts in three neighbouring houses.

(95) Jl. Hyg., Vol. XII, Pl. Suppl., p. 211; *ibid.*, vol. XIV., p. 710.

(96) Letter dated Oct. 22, 1923.

(97) Campbell Highet, Saigon Conf. Rep. 1913, p. 223.

(98) *Ibid.*

(99) *Ibid.*, p. 213.

(100) New York Med. Jl., 1912, Aug., pp. 419-420.

(101) Saigon Rep., p. 244.



Plague in Siam and Annam is mainly due to rats<sup>(102)</sup> but the shrews (*crocidura murina*) which are infested with *X. cheopis* also play a role<sup>(103)</sup>. Mendelson<sup>(104)</sup> while noting the absence of the pneumonic type in man found that a number of cats dying suddenly in one locality, suffered from pneumonic plague.

### 3. JAVA.

We are indebted to the Dutch East-India Plague Prevention Service for a complete set of their reports and to the Director (Dr. Otten) for much additional information. The data from these sources may be summarised thus:

Year	Total pl. cases	Pneu-mon.	Percent	Remarks
1911	2159	51	2.4%	Practic. all pn. c. in the Malang Dept. where pl. was first recognized.
1912	2276	10	0.43	All 10 c. in Malang.
1913	11386	23	0.20	Practic. all pn. c. in Malang.
1914	15758	563	3.60	do
1915	6237	489	7.80	Pn. c. practic. confined to Malang and 2 adj. depts. One localised outbr. (99 c.)
1916	1188	56	5.0	Primary lung plague only in Madioen and Soerabaja.
1917	421	63	14.0	2 small pneum. epidemics: Island of Madoera (25 c.); Ponorogo, Madioen (29 c.)
1918	734	44	6.0	31 c. in Ponorogo (contin. of pn. outbr.)
1919	2954	57	2.0	46 of the pn. c. in Dept. Temanggoeng.
1920	9152	162	1.7	3 small foci: Petjaloekan, Bangil (13 c.); Madioen (4 c.); Tosari, Pasoreran (4 c.)
1921	9763	620	6.4	Apparent higher incidence of pneum. type to be explained by special care taken to detect such cases (Otten, 105.)
1922	10956	939	8.5	

It can be seen from this summary that the pneumonic type is not infrequent in Java and tends to create foci when not checked in time.

The outbreak in April-May 1915 on the Boereng hills is described in detail by van Loon<sup>(106)</sup>. It arose locally from bubonic cases showing secondary lung complications. Van Loon mentions 2 probable recoveries of pneumonic cases where

(102) Cadet, Bull. Soc. Path. Exot., 1917, pp. 41-65.

(103) Kerandel, *ibid.*, 1915, pp. 54-57.

(104) Jl. Am. Med. Ass., 1919, April, pp. 1199-1205.

(105) Private information fr. Dr. Otten.

(106) Serv. Rep. for 2nd quarter 1915, pp. 35-48.

serum had been given. The administration of serum for prophylaxis seemed advantageous:

out of 68 contacts injected were 7 deaths  
 ,, ,, 15 refusing injection ,, 10 ,,

The outbreak in Ponorogo (1917-18) was also of local origin<sup>(105)</sup>; the source of the Tosari cases (1920) could not be traced. The other pneumonic outbreaks in the above table were not of local origin but arose from imported bubonic cases developing secondary lung plague. This mode of importation of pneumonic plague apparently plays an important role in Java. Soerabaja especially was such a spreading centre, but most often the cases remained isolated<sup>(105)</sup>.

The climatic conditions prevailing in Java are summarised by Otten<sup>(107)</sup>:

“Unlike British India which has pronounced climatic variations, Java enjoys a more uniform equatorial sea-climate. In the low plains the average daily temperature is 26-27° C., which in the hot months between the monsoons may rise to 28-29° C. at most. In these months the relative humidity is lowest, but seldom declines below 65%. In mountain districts at an elevation of 1500-2000 feet the mean temperature is 23-25° C. and the variations are still less, whilst the relative humidity is higher, 70% in the dry season being the minimum.”

Plague in Java seems to show a preference for mountain districts, “where the climatic conditions are more favourable for the flea.”

Bubonic plague in Java is due to rat epizootics occurring mostly among the common house rat (*Mus rattus griseiventer*). *Mus concolor Blyth* is also of epidemiological importance<sup>(107)</sup>. The question, how far the field rat (*Mus r. diardii Jentink*) is responsible for the spread of plague in Java, has attracted considerable attention, though it is doubtful if it does play an active part<sup>(107)</sup>. Besides *X. cheopis* (the main transmitter of plague in Java) *Pygiopsylla ahalae* also plays a part.

#### 4. CHINA PROPER.

Pneumonic plague has never been reported in *Yunnan*. All available records are silent in regard to the lung type. Dr. Gordon Thompson informed us in 1923 he had not heard of cases in that region. Missionary J. Graham who has lived

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(107) Transact, 4th Congr. Far East. Ass. Trop. Med., pp. 617-628.



in that province over thirty years wrote: "The plague is evidently bubonic as the Chinese speak of there being swellings in the armpits and groin with death from a few hours to a couple of days."

Similar conditions seem to prevail in Hongkong. It is true that Lawson<sup>(108)</sup> saw occasionally hemoptysis at the beginning of the present pandemic. Aoyama<sup>(109)</sup>, however, never observed copious hemoptysis and only once a case of pneumonia. Similar results were reported by Wilms<sup>(110)</sup>.

Mueller<sup>(111)</sup> summarising all possible records concluded:

"So ist wohl nur das eine anzunehmen, dass die Epidemie in Bombay, die erstarkte Palipest frueherer Jahrzehnte, durch die besonders haeufige Affektion der Lungen dem Schwarzen Tode des Mittelalters sich an die Seite stellte." (Thus it must be declared that the Bombay epidemic, more virulent than the Pali plague of previous decades, became similar to the Black Death through the frequency of pneumonic affections). In later years the pneumonic type, though occurring sporadically, never led to a serious outbreak. Some anxiety was felt in 1912, when besides secondary lung inflammations "a few undoubted cases of primary plague pneumonia did occur, one of them being a valued ward attendant in the Tung Wah Hospital"<sup>(112)</sup>.

A patient with pulmonary symptoms in April 1898 deserves special mention. He coughed onto the face of a sister on April 20. The latter fell sick on the 25th with signs of pneumonic plague and succumbed on the 29th. Another sister who attended her felt unwell on evening of May 2. (When she was admitted into hospital next morning, B. P. was present in her sputum and she died on May 5. A third sister, taken sick on May 2, and showing plague bacilli in her sputum, recovered. She had had plague in 1896<sup>(113)</sup>).

It is hardly necessary to state that domestic rodents are responsible for the Hongkong plague outbreaks. The most common fleas infesting them are *Ctenopsylla musculi* and *X. cheopis*. Occasionally *P. serraticeps* and *C. fasciatus* are found on rats<sup>(114)</sup>.

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(108) Rep. on 1894 Hongkong Epid., Hongkong, 1895.

(109) Mitt. d. Med. Fak. Tokio, 1895, Vol. III. No. 2.

(110) Hyg. Rundschau, 1897.

(111) L. c., p. 37.

(112) Hongkong Rep. 1912, p. 24.

(113) Lancet, May 26, 1900, p. 1509; June 11, 1898, p. 1635.

(114) Hongkong Rep. 1906, p. 45-47.

Pneumonic plague seems rare in *Canton* from available records and a personal communication from Dr. J. Allen Hofmann, Hacket Medical School, whose experience reaches back to the year 1908.

That pneumonic outbreaks do occur in China Proper may be gathered from the following:

- a. Simpson<sup>(115)</sup> mentions a very fatal 'influenza' epidemic raging in *Macao* in winter 1894-95 and suspects it to have been pneumonic plague.
- b. During the plague in the spring of 1900 at *Hoihow* (Hainan) many pneumonic cases were noted. Up to the end of June 5700 persons died in the town of 30,000 inhabitants<sup>(116)</sup>.
- c. In February 1913 pneumonic plague was seen in several localities round *Amoy*. Earlier in 1910 a foreign physician had contracted this type of infection from a Chinese patient suffering from the same<sup>(117)</sup>.
- d. About 14 pneumonic cases occurred among close contacts in April 1915 in *Swatow*<sup>(118)</sup>. The infection had apparently been introduced from one of the plague affected villages in the district<sup>(119)</sup>.
- e. More difficult to determine is an outbreak reported in 1922 from *Foochow*, involving two English missionary doctors. Plague had prevailed in Foochow since the middle of April, 1922, but as Dr. Marion Hook informs us, it was of the bubonic and septicemic types; pneumonic plague, generally rare in Foochow, was not seen. At the beginning of June one of the hospital cooks fell sick; she went to her village, but returned to the hospital on June 8 in a serious condition. Dr. Lawson examined her and diagnosed pneumonia. She died after a few hours. It was also stated that two of her relatives in her village died with similar symptoms. Four days after the death of the cook (12th) Dr. Lawson was taken ill suddenly with high fever and bilious vomiting. He complained of severe pains in the left side of his chest, where a pneumonic focus developed. On the 13th large quantities of thin bloody sputum were expectorated. The

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(115) P. 64.

(116) L. G. B. R. 1898-1901, p. 346.

(117) Ibid., 1913, p. 31; 1910-11, p. 147.

(118) Ch. Med. Jl. 1917, p. 33.

(119) L. G. B. R. 1914-17, p. 86.



doctor died on the 14th. He had been attended by Drs. Hook and Mackenzie and—for a short time—by an English nurse; none of them had any mask on. Dr. Mackenzie was attacked with similar symptoms four days after Dr. Lawson's death and succumbed in less than sixty hours; he was attended solely by Drs. Hook and Fisher, who then used masks when visiting him.

The Shanghai Municipal Laboratory, where slides were sent, reported that "the sputum contained bacilli, but appearances are not sufficiently characteristic to establish a diagnosis of pneumonic plague. Most of the slides were broken." A similar report was made by the Hongkong laboratory. Dr. M. Hook who attended the patients diagnosed plague bacilli in their sputum.

The outbreak seems rather suspicious. Pneumonic cases are occasionally seen in Foochow. Thus Dr. Hook attended 4 cases (all fatal) in one family in July, 1921, strongly suspicious of pneumonic plague.

- f. During the outbreak of bubonic plague in the mining village of Tongshan (Chihli Prov.) in 1908, the number of pneumonic cases seen was considerable. Gray<sup>(120)</sup> noted that "at the beginning, bubonic cases greatly outnumbered the other varieties, but septicemic and pneumonic cases were met with in increasing frequency. About 86% were bubonic, 12% pneumonic and 3% septicemic." According to our investigations, the epidemic (1000 cases with 800 deaths) began at the end of August and finished towards end of November. The source of the epidemic could not be definitely determined, but Andrews investigating the rats from June, 1909, to September, 1910, found that the flea rate was high in autumn. He noticed only *M. decumanus* and *X. cheopis*<sup>(121)</sup>.

## 5. SOUTH MANCHURIA.

Up to 1910 plague outbreaks in this province seem to have been imported by rats from the endemic south. From then onwards the history of the disease is closely associated with the tarabagan (marmot) of Siberia and Mongolia.

The first epidemic seen was in 1899 and was mainly bubonic; secondary lung involvement and skin hemorrhages

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(120) Pl. Outbr. in Manchuria and South China, Peking 1911, p. 14.

(121) Mukden Conf. Rep., p. 59.

(up to size of a Chinese dollar) were also noted<sup>(122)</sup>. The outbreak terminated on Nov. 13. After a clear interval it appeared again at the beginning of December, when a whole family was exterminated by pneumonic plague except the mistress of the house. Two weeks afterwards she went to live with some relatives. Within five days plague appeared in this second house, where six cases (2 pneumonic, one with inguinal bubo and secondary pneumonia, and 3 bubonic) were noted. Four out of these, including the woman who was alleged to be the carrier of infection, died<sup>(123)</sup>.

A second outbreak in Newchwang from August 1901 to Jan. 1902 was purely bubonic.

In September 1902 some pneumonic cases were observed at Kaichow and adjacent places by the Russian doctors. It appears that this type had been present together with the bubonic early in the same month<sup>(124)</sup>. Little more is known about this epidemic which killed 250 persons.

Pneumonic plague was absent in the 1903, 1905, 1906 epidemics.

In 1907 (January) 23 pneumonic cases were seen in Newchwang and adjacent villages. The disease reached a group of villages about 8 E. miles from Newchwang where 80 people succumbed to pneumonic plague up to February. In the second half of 1907 only the bubonic and septicemic types were noted<sup>(124)</sup>.

The rats in South Manchuria were examined at the time of the 1910-11 epidemic. While Petrie found only *M. decumanus* among a few hundred rodents examined by him, infested with *X. cheopis* and *Ceratophyllus* sp., Kitasato reported among the 30,000 rats examined by Japanese doctors in South Manchuria *M. rattus* was found in 6%. Out of 127 fleas examined in Mukden 48 were *X. cheopis*, 79 *Ceratophyllus anisus*<sup>(125)</sup>.

## 6. FORMOSA.

Primary plague pneumonia was absent in the early invasions of plague in Formosa. Yamigawa saw only 'metastatic foci' in the lungs<sup>(126)</sup>.

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(122) Avrorov, Pl. Rep. of Ch. E. Railw., Harbin, 1912.

(123) Klodnitzki, R. Vrach, 1905, pp. 1595-99.

(124) Pl. R. Ch. E. Rw., Harbin, 1912, Suppl., pp. 1-33.

(125) Mukden C. Rep., pp. 60-61.

(126) Virchow's Arch., Vol. 149, p. 54, 92, Suppl.



Takaki<sup>(127)</sup> later on tabulated the different types of plague met with in 1898-1908 in the four isolation hospitals at Taihoku, Tainan, Keelung and Kagi as follows:

<i>Type.</i>	<i>Cases.</i>	<i>Deaths.</i>	<i>Mortal%</i>	<i>Incidence%</i>
Bubonic .....	2571	1557	60.56	97.16
Pneumonic ....	50	48	96.00	1.89
Intestinal .....	2	2	100.00	0.075
Carbuncle ....	8	5	62.50	0.300
Eye plague ....	1	1	100.00	0.037
Septicemic ..	14	7	50.00	0.518
Total .....	2646	1620	61.22	—

The author mentions two separate outbreaks of pneumonic plague, viz.

- a. in the Kinkaseki Gold Mines near Keelung (6 c.)
- b. in two villages of the Shoka district (12 c.)

As no epizootics could be found among local rats and mice it can be assumed that the infection was imported through human agency.

Both Takaki and Kuraoka<sup>(128)</sup> agree that *M. rattus*, *M. decumanus* and *M. musculus* were responsible for the human outbreaks. *Mus agrarius* and a 'kind of field mouse' were found experimentally susceptible, but no natural infection was noted among them. In the Kagi district many rabbits died during one human outbreak but the cause of their deaths was not ascertained. The *Loemopsylla cheopis* flea was the principal means of spread.

## 7. JAPAN.

Pneumonic plague occurred frequently during the first serious outbreak in *Kobe* and *Osaka* (Nov. 1899 to Jan. 1900). The Official Report<sup>(129)</sup> gives a total of 69 cases with 63 deaths and describes only one pneumonic focus which started from a female worker in a cotton factory in Osaka. To this case could be traced the infection of 18 persons. Five coworkers developed bubonic plague and were supposed to be infected from the sputum expectorated by the woman on the floor of the factory. The authors remark that this woman was sitting opposite a wall while at work and for this reason no pneumonic cases

(127) D. Hyg. Verh. d. Insel Formosa, Dresden 1911.

(128) Saigon Conf. Rep., pp. 206-207.

(129) Kitasato, Takaki, Shiga and Moriya, Ber. ueb. d. Pestepid, etc., Tokio, 1900.

occurred in the factory. The remaining 13 cases were pneumonic and included three doctors and members of their families. The wives of two doctors fell sick, when attending their husbands, though they had received prophylactic serum treatment. This pneumonic outbreak is frequently mentioned as the only one which ever occurred in Japan.

It would seem that both the total number of plague cases and the pneumonic ones were higher than the figures in the official report. At a meeting of the Central Sanitary Association at Tokio in December<sup>(130)</sup> it was stated that in September and October 1899 (i.e. before the presence of plague had been diagnosed) 230 deaths attributed to 'Acute Meningitis' had occurred at Kobe as well as 266 diagnosed as 'Beri-Beri' and 270 certified as 'Acute Pneumonia'. Thus Kitasato and Ogata were of opinion "that most of the above cases were in all probability plague, and that, therefore, the deaths due to the epidemic.....must have numbered some hundreds."

Similarly it was stated that the number of plague cases at Osaka was far above the official figures and that many were of the pneumonic form. Data concerning the frequency of pneumonic plague in Osaka are given in a paper of Masuyama<sup>(131)</sup>. He observed during 1899-1907 383 patients out of a total of 974 cases with 880 deaths, the varieties being:

<i>Year.</i>	<i>Bubon.</i>	<i>Pneum.</i>	<i>Skin pl.</i>	<i>Septic.</i>	<i>Larynx pl.</i>	<i>Eye pl.</i>
1899-00	61	12	1	2	—	—
1905	56	1	—	—	—	—
1906	60	1	—	2	1	—
1907	158	10	1	16	—	1
	—	—	—	—	—	—
Total	335	24	2	20	1	1

Masuyama remarks that pneumonic plague most often attacked medical men and the attendants who had to carry patients into the Hospital. The following information of plague deaths among medical men in Japan is interesting:

In Osaka 4 from pneumonic and 1 from laryngeal pest.

Two of these passed on the infection to their families.

In Kobe 1 from pneumonic

In Sumoto 1 „ bubonic

In Tokio 1 „ eye plague.

Shibayama<sup>(132)</sup> reported 8 lung cases out of a total of 153 at Kobe from August 1905 to Dec. 1906. Five of these received serum therapy with the following results:

(130) L. G. B. R. 1898-1901, pp. 366-367.

(131) Z. f. Klin. Med., Vol. 70, No. 5 & 6.

(132) Mukden C. Rep., pp. 125-126.



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|----|--|----------------|-----------|--|
| 1. |  | Prophyl. ....  | 20 c. c.  | Died 8th day.                                |
|    |  | Therapeut. ... | 400 c. c. |  |
| 2. | Coolie, disinfect. plague houses.            | Therap. ....   | 320 c. c. | Recovd., having shown B. P. in his sputum.   |
| 3. | Doctor, prob. infect. when att. c. of abort. | Therap. ....   | 240 c. c. | Died 3rd day.                                |
| 4. | Woman att. plague patient.                   | Prophyl. ....  | 40 c. c.  |  |
|    |  | Therapeut. ... | 240 c. c. | Recovered. Sputum?                           |
| 5. |  | Therap. ....   | 80 c. c.  | Died soon after adm. on 6th day of sickness. |

Two patients thus recovered.

Another pulmonary outbreak occurred in July 1914 in the province of *Kanagawa*<sup>(133)</sup>.

Kitasato found no direct connection between the pneumonic outbreaks and rat epizootics<sup>(134)</sup>.

The rats of Japan are *M. rattus*, *M. decumanus*, *M. alexandrinus*<sup>(135)</sup>.

Kitasato believes that although *ceratophylli* (*C. anisus* and another species) are the most prevalent of fleas in Japan—plague is mainly spread by *Loemopsylla cheopis* imported from India<sup>(135)</sup>.

#### IV. PNEUMONIC PLAGUE IN THE ISLANDS OF THE PACIFIC AND IN AUSTRALIA.

##### 1. PHILIPPINES:

When plague was introduced into the islands in 1900 many cases were not properly certified<sup>(136)</sup>. In January 1900 not less than 212 deaths were ascribed to eclampsia. 'Respiratory diseases' and 'tubercle of lungs' also figured prominently but it is not certain how many of these were pneumonic cases.

Lung symptoms were rare and of a secondary nature in the later years<sup>(137)</sup>.

##### 2. AUSTRALIA.

Very few cases are reported from Australia. A small group was noted in June 1906 at Balmain, a suburb of Sydney, apparently connected with mild plague cases occurring in the city and neighbourhood<sup>(138)</sup>.

(133) L. G. B. R. 1914-17, p. 91.

(134) Z. f. Hyg., 1909, pp. 279-284.

(135) Phil. Jl. Sc., 1906, June, p. 477.

(136) L. G. B. R., 1898-01, p. 379.

(137) Schoebl, Ph. Jl. Sc., 1913, Dec., p. 415; Goff, Jl. Am. Med. Assoc., 1913, June 28.

(138) L. G. B. R. 1906-07, p. 93.

## 3. NEW CALEDONIA.

Plague became publicly known in this French penal settlement in the Pacific Ocean in Dec. 1899, but it had undoubtedly existed in the preceding month when several natives died from 'tuberculosis.' During the later stages of the epidemic lasting up to the end of April 1900, pneumonic cases were not uncommon. "Some of these among the white population were ascribed to inhalation of infective dust while employed in pulling down houses in which plague cases had previously occurred"<sup>(139)</sup>.

During the 1912 outbreak some pulmonary and septicemic cases were reported together with 50 bubonic ones<sup>(140)</sup>.

## 4. HAWAIIAN ISLANDS.

As elsewhere the first officially recognised plague outbreak in Honolulu (Dec. 12, 1899) was complicated by an increase in the mortality ascribed to other causes. Pneumonic plague might be partly responsible for this increased fatality as the presence of "pneumonia with a high rate of mortality" was already recorded in December 1899<sup>(141)</sup>.

In later years, lung cases seemed infrequent. Thus we found two (one Chinese and one Japanese) reported in 1910; two (both Japanese) in August 1922<sup>(142)</sup>.

The rats inhabiting the islands are *M. alexandrinus* and *M. rattus*; the latter sometimes in the country<sup>(143)</sup>.

## V. PNEUMONIC PLAGUE IN AMERICA.

## 1. SOUTH AMERICA.

Our information about South America is rather vague because plague outbreaks were often not at all or incompletely recorded. While plague is often mild in character a number of pneumonic outbreaks may be quoted:

## I. Brazil:

A. One raging in San Paulo City from Dec. 1899-Feb. 1900. The disease was apparently imported from Santos, causing sporadic deaths in November 1899, and increasing in

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(139) Ibid., 1898-01, pp. 389-390.

(140) Ibid., 1912-13, p. 49.

(141) Ibid., 1898-01, pp. 385-386.

(142) Ibid., 1910-11, p. 154; U. S. P. H. R., 1922.

(143) L. G. B. R., 1913, p. 35; McCoy, Am. Jl. Hyg., 1921, March, p. 187.



numbers in December, both among the rich and poor. No figures were published<sup>(144)</sup>.

B. Two suspicious outbreaks of (a) 'virulent influenza' at Rio Grande do Sul (1900-<sup>145</sup>) and (b) 'tuberculosis' at Rio de Janeiro (1900-<sup>146</sup>).

C. One observed by Rau<sup>(147)</sup> in Santa Maria, in the province Rio Grande do Sul, in 1912.

It appears that this inland locality had been visited by plague during the four previous years; the rats had been attacked and the epizootic caused sporadic human cases with 50% mortality.

The 1912 epidemic was also preceded by an epizootic, one of the first victims being a man living in an insanitary bakery where 200 dead rats were found. This man developed at end of July pneumonic plague and passed it onto 16 others. A 17th case developed cervical buboes and pneumonia. All the patients died, though serum was administered to them. None of the staff were infected, being protected by masks. A German priest was among the victims.

The outbreak occurred during the cool season and the houses were not artificially heated<sup>(148)</sup>. This statement is important though it should be pointed out that another city (San Paulo) showed pneumonic plague at another period of the year.

D. One suspicious outbreak at San Paulo, first taken for influenza but later regarded as pneumonic plague. Suspicious bacilli, corresponding in almost all respects to the B.P., were found both in human beings<sup>(149)</sup> and in wild rats<sup>(150)</sup>.

## II. *Paraguay.*

Some fatal plague cases in Asuncion in autumn 1899 were certified as 'pneumonia' and seemed suspicious. In later years actual plague pneumonia was recorded: Lindsay<sup>(151)</sup> notes in 1907 groups of such cases in a town near Asuncion

(144) L. G. B. R. 1898-01, pp. 436-437; 1899-00, p. 340.

(145) Ibid., 1899-00, p. 341.

(146) Ibid., 1898-01, p. 439.

(147) D. Med. W., 1912, No. 49, pp. 2314-2315.

(148) Teague, Ph. Jl. Sc., Vol. VIII b, p. 243.

(149) Cintra, Bol. Soc. Med. e Cir. de S. Paulo, 1920 & 1921, Oct.—Feb., pp. 358-61.

(150) Smillie, Jl. Inf. Dis., 1920, Oct., pp. 378-384.

(151) B. M. Jl., 1911, Nov., p. 1329.

and again in August and Sept. 1911 in Asuncion and Villa Concepcion. In the last two outbreaks three doctors and a priest died.

### *III. Columbia.*

Cases of 'infectious pneumonia' were seen in different localities during 1914. Their plague nature was sometimes denied, but in other instances the B.P. was diagnosed in the sputum of the patients<sup>(152)</sup>.

## 2. NORTH AMERICA.

### *I. Mexico.*

An outbreak called 'epidemic pneumonia' occurred in May 1904 near Mazatlan (State of Sinaloa), a town visited by plague in 1902 and 1903<sup>(153)</sup>. Though details were scarce it was probably pneumonic.

### *II. U. S. of North America.*

In our paper on "Plague in Wild Rodents" we recognised two groups of epizootics. In the first rats were infected at sea-ports, leading to human outbreaks. Later on the infection passed from the rats to the wild rodents of California, then ran its course among them and finally produced in human being a clinically different disease<sup>(154)</sup>.

A pneumonic outbreak caused directly by rats has never been observed in U. S. Even sporadic cases of pneumonic plague, arising from them are rare<sup>(155)</sup>. The only pneumonic epidemic recorded in U.S. originated in the wild rodents. Here, the first victim—the story reads familiarly to us, who are used to the tarabagan—caused outbreaks in Transbaikalia—hunted in the fields in August 1919 and probably brought home a squirrel to prepare for food. This man fell sick four days afterwards, developing an axillary bubo and secondary pneumonia. He infected 13 other persons, including two doctors. This pneumonic outbreak occurred, when the weather was warm and dry<sup>(156)</sup>.

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(152) L. G. B. R. 1914-17, p. 99; Lancet 1914, July, p. 110.

(153) L. G. B. R. 1904-05, p. 264.

(154) Mc. Coy, Am. Jl. Hyg., 1921, March, p. 187, 191.

(155) Kinyoun, Jl. Am. Med. Assoc., Vol. XLII, No. 3; Hartman and Bowie, *ibid.*, Vol. 78, p. 493.

(156) Kellogg, Am. Jl. Publ. H., 1920, p. 599; Harrison, Mthly. Bull. Cal. St. Dept. Agric. 1920, pp. 187-194.



## VI. PNEUMONIC PLAGUE IN AFRICA.

## 1. EGYPT.

We are indebted to the Egyptian Public Health authorities for a set of their plague reports and two Progress Reports.

There seems to be no mention of pneumonic plague in Egypt before the beginning of the present pandemic. This absence was significant during the first half of the 19th century, when the Egyptian plagues were carefully observed by Clot-Bey<sup>(157)</sup> and other observers. It must be assumed that this type was present in historical times, especially during the Black Death, but the recent Egyptian Plague Report<sup>(158)</sup> remarks that "they have failed to discover in the early writings any indication that pneumonic plague was recognised as a distinct variety of the disease."

Gotschlich observed<sup>(159)</sup> in Alexandria (1899) 91 bubonic cases (5 with secondary pneumonia), one septicemic case and 4 primary pneumonic cases. Gotschlich also reported recovery in two patients with secondary lung involvement and one with primary pneumonic signs, claiming that in the sputum of those three patients the B.P. was present for a prolonged period of their convalescence.

In 1900 seven men engaged in disinfecting houses contracted plague, two in the pulmonary form<sup>(160)</sup>.

It is doubtful if this type was present among the early cases in Port Said (spring 1900) when only 'infectious influenza' and 'pneumonia' were reported<sup>(161)</sup>.

Sticker<sup>(162)</sup> recorded a pneumonic epidemic starting at the end of 1900 in Tantah (Lower Egypt) among negroes and Bedouins, which was spread into the villages of Benha and even to Upper Egypt after the expulsion of the Bedouins.

Gotschlich<sup>(163)</sup> reviewing in 1903 the plague epidemics of the preceding years, distinguished between a *summer type* which was often of an exclusively bubonic character with a low mortality and a *winter type* which showed reverse features. Among 105 winter cases were 38 with primary plague pneumonia and 10 with "primary hemorrhagic plague

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(157) Quot. by Mueller, l. c.

(158) Cairo, 1923, p. 4.

(159) Z. f. Hyg., Vol. 35, 1900.

(160) U. S. P. H. R., Aug. 10, 1900.

(161) L. G. B. R., 1898-01, p. 142.

(162) Vol. I, p. 380.

(163) Festschr. f. R. Koch, Jena 1903, p. 541.

sepsis." The figures from 1904 onwards for the Delta and Northern and Southern Upper Egypt are as follows:—

Year	Delta		N.U.E.		S.U.E.		Remarks
	Bub. and Sept.	Pneum.	Bub. and Sept.	Pneum.	Bub. and Sept.	Pneum.	
1904	277	11	270	0	276	20	1 pneum. outbr. in the Delta 4 " " in S.U.E.
1905	243	20	2	0	1	0	
1906	190	20	106	36	83	196	17 " " in S.U.E.
1907	276	13	413	47	312	192	2 " " in N.U.E.
1908	539	16	562	93	242	59	15 " " in S.U.E.
							1 " " in the Delta
							2 " " in N.U.E.
1909	297	3	202	5	5	1	15 " " in S.U.E.
1910	418	1	584	30	77	128	4 " " in N.U.E.
1911	193	3	387	38	898	127	5 " " in S.U.E.
							1 " " in N.U.E.
1912	197	6	433	20	140	88	11 " " in S.U.E.
							1 " " in the Delta
							1 " " in N.U.E.
1913	310	9	277	25	15	18	7 " " in S.U.E.
							1 " " in N.U.E.
1914	112	1	79	3	3	21	88 pneum. outbr.
1915	38	0	163	34	0	0	
1916	121	3	1463	58	22	35	
1917	366	19	224	14	83	26	
1918	20	2	301	4	2	28	
1919	206	3	520	8	75	65	
1920	214	7	198	22	17	4	
1921	289	5	50	1	4	7	
1922	246	1	222	7	9	2	

The Egypt Report 1923 emphasizes the high incidence of pneumonic outbreaks in Qena and Girga (S.U.E.). Their origin may be traced throughout Egypt to plague sufferers with secondary lung features. The report lays stress upon the role of plague sick travellers who are liable to develop such symptoms and introduce the infection to their places of destination.

Most of the pneumonic outbreaks in Upper Egypt which form the material of the 1923 report, occurred during the hot and dry plague season. Outbreaks during the off-season are not frequent and are due to plague sufferers who "travel from the Delta when the disease is prevalent there."

No recovery from the primary type was recorded; the prognosis of secondary lung affection was declared serious, but not hopeless.



In sixty purely pneumonic outbreaks women formed 71% of the sufferers, a fact well in accord with the familiar character of lung plague in Upper Egypt. The greatest morbidity lies between 20-50 years and is due to "the degree of exposure" and not to "any specific difference in susceptibility."

Cases among 'barbers' on hospital duty and female corpse-washers are also mentioned.

Besides *M. rattus* and *Mus norvegicus* the role of *Arvi-canthus niloticus* is now confirmed. *X. cheopis* is the principal flea seen in all three species.

The report concludes that "an unusual degree of susceptibility of the inhabitants mainly accounts for the heavy incidence of pneumonic plague in the Qena-Girga district and that it is heightened by the exceptional aridity which prevails there in the plague season."

## 2. ALGERIA.

Passing from Egypt to Algeria, we must remark upon the apparent absence of pneumonic plague in the old focus of Cyrenaica.

In Algeria plague pneumonia was observed by Benoit<sup>(164)</sup> in August 1903. This was not preceded by a rat epizootic and the infection was thought to have been recently imported by a grain ship from Egypt. The first victim was a young girl, who had worked in a mill in Algiers. She became sick, developing apparently a tonsillar affection with cervical buboes and secondary pneumonia. When ill, she went home to her family in Blida, 48 km. away and infected her parents. All three died.

A second outbreak of pneumonic plague, occurring in Ruisseau, a suburb of Algiers, deserves attention<sup>(165)</sup>.

Between July 1-7, 1912, three members of a family and their servant succumbed to pneumonic plague, first a boy, aet. 14, who used to stay away from school and hunt rats with his rifle. Two more cases developed on July 12: (a) in the aunt of the boy who had nursed her relatives and (b) in a woman who had attended the servant. Both had received prophylactic serum injections, but were at that time in the incubating stage. The first patient died after one day, the second on the 5th, having received large serum doses intravenously and subcutaneously.

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(164) Arch. de Med. et Pharm. Milit., 1904, Febr., p. 97.

(165) Raynaud. Rev. d'Hyg. et Pol. San., 1912, Aug., pp. 861-867; Lemaire, Rev. d'Hyg., 1912, p. 1219.

It would seem that this outbreak was preceded by an epizootic, though no trace of plague was found among the 1610 rats examined after the outbreak. However, the inhabitants had noticed a diminution in the rat population before the onset. Two out of three cats also died after an illness of 2-3 days before the onset.

Raynaud adds: "La forme pulmonaire s'est établie d'emblée; aucun n'a présenté de bubons" (the pneumonic type was present from the very start; none of the attacked had buboes).

### 3. MOROCCO.

The only notes in our possession refer to a pneumonic outbreak in the Jewish quarter of Tangier early in October 1911. The population of the invaded houses was evacuated to a camp three miles outside the town<sup>(166)</sup>.

### 4. FRENCH WEST AFRICA.

Plague was introduced into Dakar, the capital of French West Africa, in April 1914, most probably by the steamer "Mingrelie" from Casablanca (Morocco)<sup>(167)</sup>. The epidemic lasted up to Jan. 15, 1915, spread to other places in the colony and the Cape Verde islands, and claimed almost 9000 victims. The village Yoff, 16 km. from Dakar suffered badly, only one fifth of a population of 1200 being spared<sup>(168)</sup>. All reports on this outbreak<sup>(169)</sup> agree upon the prevalence of the pneumonic type which was evident at the beginning when no epizootic was present. The first infected rats were found in July, 2½ months after the onset and from thence onwards the bubonic type predominated.

Two more items of interest may be added:

- A. Rousseau<sup>(170)</sup> referring to some pneumonic cases at N'Diourbel in summer 1914, stated that "some of the white contacts were found to have agglutinated plague bacilli in the sputum, while showing no evidence of disease." We have not seen the original paper and are therefore not sure if those were healthy carriers or what tests were employed.

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(166) L. G. B. R. 1912/13, p. 87.

(167) Kermorgant, Bull. Acad. Med., 1916, Aug. 22, pp. 126-133.

(168) Lafont, Bull. Soc. Path. Exot., 1915, Nov., pp. 660-680.

(169) Marcandier, Arch. Med. & Pharm. Nav., 1918, Aug. & Sept., pp. 125-145 & 191-219.

(170) Paris Med., 1917, Dec., quot. fr. Trop. Dis. Bull. Vol. 12, p. 407.



B. Several instances of illness in domestic animals and animals kept in captivity were suspected to be plague but actual proof was obtained only in two cats, a monkey and a civet cat<sup>(171)</sup>. Kermorgant emphasized the dangerous role which cats might play in the propagation of the disease.

Plague did not cause much havoc up to the year 1918, when it claimed almost 3000 victims, being present throughout the year and most prevalent in May-September.

The number for 1919 was still higher, over 4000 cases being counted in the colony and 712 in Dakar. The worst period was July-October. In Dakar were<sup>(172)</sup>:—

Bubonic            63%    Pneumonic        30%    Septicemic    7%.

The epidemic reached an abrupt end in December, at which time the rats had almost disappeared<sup>(173)</sup>.

Huchard<sup>(174)</sup> summarizes the plague cases occurring in Dakar in 1920 and 1921 as follows:

<i>Type.</i>	<i>1920</i>		<i>1921</i>	
	<i>Certain</i>	<i>Probable</i>	<i>Cert. c.</i>	<i>Prob. c.</i>
	<i>cases.</i>	<i>cases.</i>		
Bubonic .....	24	1	323	2
Pneumonic .....	13	23	11	7
Septicemic .....	4	8	38	116
Unclassified .....	1	7	230	175
Total .....	42	39	602	300

From October 1, 1921 to Oct. 1, 1922 were registered in Dakar altogether 93 cases, 76 being treated in the Hôpital Indigène d'Instruction. Lhuerre<sup>(175)</sup> gives the following details about the 76 admissions:

<i>Type.</i>	<i>Record.</i>	<i>Died.</i>
Simple bubon .....	28	—
Complic. bubon .....	3	1
Pneumon. ....	19	6
Septic .....	7	3
Within the first 24 hours died.		
Pneum. ....	—	6
Others .....	—	3
Total .....	57	19

(171) L. G. B. R. 1914/17, p. 117.

(172) Esquier, Arch. Med. & Ph. Nav., 1920, Sept., pp. 187-213.

(173) Noc., Rapp. sur le fonct. de Lab. de l'A.O.F. en 1919, Dakar, 1920.

(174) Bull. Soc. Path. Ex., 1923, p. 375.

(175) Ibid., 1922, pp. 874-906.

Thus we have 31 pneumonic cases with only 12 deaths! The author, discussing this remarkable result, points out that the prognosis of pneumonic plague is to all appearances better in warm than in cold climates. The therapy used by him consisted of serum administration (100 c.c. intravenously on day of admittance, same repeated on the 2nd or 3rd day; doses being changed in individual cases) and—following Chenot's dictum "pestis non sine ratione dicitur morbus cordis" (plague is justly considered as an affection of the heart)—in a careful heart therapy (digitaline in granules; sparteine-strychnine subcutaneously; stimulants-acetate of ammonia, liquor Hoffmanni-per os).

Heckenroth states<sup>(176)</sup> the plague season in Senegal is moist and hot, adding that—though there is an appreciable difference in temperature and humidity between the coast districts and the interior of Senegal—plague shows a similar course in both localities.

The grey and black rats<sup>(177)</sup> and the domestic mice are responsible for plague outbreaks. The importance of the mice, infested with *X. cheopis* like the rats, has been recently emphasized<sup>(178)</sup>. Besides *X. cheopis* a possible role is played by the sand flea, *Echidnophaga gallinacea*. This insect is widespread and found on poultry, dogs, cats and also human beings who go barefoot or work with their hands near the ground. Noc<sup>(179)</sup> infected a white rat, harboring these fleas, and recovered B. P. from the feces of the fleas after the rat had succumbed. The fleas stick to their dead hosts for a considerable time. We may mention in passing that an insectivore (*Crocidura stampflii*) is thought to play "a part different to that of the rodents but not without importance," acting as a *reservoir* and infecting rodents through its fleas (*X. cheopis*)<sup>(180)</sup>.

## 5. GOLD COAST.

The plague outbreaks occurring in these parts may be thus summarised:

1891-92 Suspicious pneumonic.

1899 *Ivory Coast*: From March to May outbreak of 'Lymphangite infectieuse', preceded app. by rat epizootic, claiming at least 200 among the 1000 black

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(176) Bull. Soc. Path. Ex., 1923, p. 374.

(177) Noc., l. c. & Bull. Soc. Path. Ex., 1921, pp. 516-519, Teppaz, *ibid.*, pp. 514-16; Leger & Baury, *ibid.*, 1923, pp. 133-137.

(178) Leger & Baury, *ibid.*, 1923, pp. 133-137.

(179) Qu. fr. Trop. Dis. Bull., Vol. 20, p. 20.

(180) Leger & Baury, C. R. Acad. Sc., 1922, Feb., pp. 423-426.



inhabitants of Grand Bassam. Three medical men died. Suspic. outbr. in September<sup>(181)</sup>.

1907-08 Extensive pneumonic.

1909-12 No records of pl. Extensive rat campaign in 1911 showed no infected rodents<sup>(182)</sup>.

1913 Outbreak "strongly resembling bubonic plague" in the fishing village Tunga, near Yeji, northern territory of colony. 11 c. with 8 d. among 30 inhabitants<sup>(183)</sup>.

1914 Similar outbreak in adjacent fishing village, Makongo. 9 c. with 5 d. Origin of both outbr. unknown<sup>(184)</sup>.

1915 No plague found, but number of death returns attributed in Accra to 'pneumonia and cough' suspiciously high (14% out of 843 total d.)<sup>(185)</sup>.

1916 139 out of 1131 total deaths ascribed to pneumonia and cough<sup>(185)</sup>.

1917-18 Three outbreaks suspicious f. pneumon. pl.

1919-22 No records.

The outbreak in 1892 was not known at the time to be pneumonic plague. It is now evident that the 'influenza' starting at the close of 1891 and becoming malignant in 1892, was no other than pneumonic plague. Confirmation was obtained from the meteorological observations of 1891-92 which closely resembled those of 1907-17<sup>(186)</sup>. The 1907-08 epidemic is exhaustively described by Simpson<sup>(187)</sup> and by Fisch<sup>(188)</sup>. The first Accra invasion, lasting from November to April 8 was preceded by a rat epizootic<sup>(189)</sup> and was mainly bubonic. Pneumonic cases occurred occasionally during its whole course, more frequent toward the end and apparently in January also.

Outside Accra no local epizootic was discovered and a pneumonic type prevailed in addition to some cervical cases. One outbreak at Nyinyano (6 hours distant from Accra) and another at Anamaboe (26 hours distant from Accra) may be

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(181) L. G. B. R. 1898-01, p. 208; 1899-00, p. 333.

(182) Med. & San. Rep. Gold Coast, 1911.

(183) L. G. B. R., 1913, p. 48.

(184) Ibid. 1914-17, p. 118.

(185) Ibid.

(186) Gold Coast Rep. 1912, p. 93.

(187) Rep. on Plague in the Gold Coast, London 1909.

(188) Arch. f. Schiffs & Tropenh., 1908, No. 15.

(189) Paper on Wild Rodents.

described. At the former place 25 men, 37 women and 2 children died from Jan. 22-Feb. 23. The epidemic at Anamaboe could be traced to the same group of 3 men from Accra who had infected Nyinyano. Altogether 24 persons died.

Fisch remarks that in Accra the victims were almost exclusively poor people, while in the pneumonic outbreaks all classes of the population were affected.

Simpson records one recovery from pneumonic plague in a patient who had been previously inoculated with Haffkine's vaccine.

The second Accra outbreak (purely pneumonic) was detected at beginning of June and traced to a village whence an infected family had fled to Accra. The third Accra outbreak is believed by Simpson to have originated in "some clothing or other effects from an infected house."

The approximate number of cases may be summarised thus:

1. Up to April	In Accra	168 (127 d.)	Bubon. with pn.
	Outs. "	134 (131 d.)	Pncum. with bub.
2. May-June 26	In "	23 ( 23 d.)	Pneumon.
	Outs. "	17 ( 17 d.)	"
3. July 28-Aug. 17	In "	6 ( 6 d.)	"
4. Oct. 13	In "	1 ( 1 d.)	"
Total		349 (305 d)	

Among the measures adopted to suppress the epidemic were:—

Isolation of contacts and inoculation with Haffkine's prophylactic, evacuation of some badly infected areas, establishment of a land cordon which could be passed only by persons vaccinated at least a week before. While Simpson praises the cordon, Fisch doubts its efficiency.

In February 1917 some suspicious cases occurred at Ofako, 9 miles inland from Accra. Early in March similar cases were observed in Accra where the diagnosis was confirmed. Among the six cases seen in Accra, the first (girl, aet. 15) had an axillary bubo, while the next (a child, aet. 5) had apparently septicemic plague. A total of at least 39 cases was reported, mostly of the pneumonic type<sup>(190)</sup>.

9 cases with 7 deaths, said to have been pneumonic, were noted in December 1917 at Axim, a port 150 miles west of Accra. Suspicious cases were again observed in the locality in July 1918<sup>(191)</sup>.

(190) Gold Coast Rep. 1917.

(191) L. G. B. R. 1914-17, p. 119.



The climate of the colony is stated<sup>(190)</sup> to be damp and hot, though cooler than in other similarly situated tropical countries, especially at night. The rainy seasons, accompanied by a considerable fall in temperature, last from March to July and from September to October. The years 1916 and 1917 were exceptionally wet.

## 6. CENTRAL AFRICA.

Early observers in this ancient home of plague remarked upon the absence not only of the pneumonic type but also other pneumonic affections<sup>(192)</sup>. Plague in that region was believed to be milder than in India and this was ascribed to the absence of overcrowding and to the open air life of the natives, who seemingly realise the infectiousness of the disease and evacuate temporarily affected localities<sup>(193)</sup>. The reports at our disposal do not mention the existence of plague pneumonia up to 1903. The end of 1903 saw an outbreak accompanied by frequent lung symptoms at Iringa (then in German East Africa). This lasted until the beginning of March. Rat infection was present in addition to 47 human cases, almost all fatal. Among the victims were two sisters and a priest of a neighbouring Catholic Mission. The natives said that a similar illness appeared almost every year<sup>(194)</sup>.

There was a lull until 1909 since when it has been unusually active including the pneumonic type<sup>(195)</sup>.

Of the many outbreaks that of 1912 may be specially mentioned. This occurred in the Gasseni district (then in German East Africa). The figures are as follows:

Gasseni .....	55	pneumon.,	3	bubon.
Usseri kwa Demassi .....	11	,,	0	,,
<hr/>				
Total .....	66	pneumon.,	3	bubon.

Lurz<sup>(196)</sup> states expressly that the first case was of the *pneumonic* type and that his illness must have been due to the rats<sup>(197)</sup> because he had not been absent from his home village (Gasseni) for two months. The patient was, however, never seen by a physician.

(192) Zupitza, Z. f. Hyg., 1898, pp. 268-294.

(193) L. G. B. R. 1898-01. p. 172.

(194) Ibid. 1904-05, p. 275-276.

(195) Uganda Ann. Med. Rep., 1911, 1912; East Africa Prot. Ann. Rep. 1911, 1913; Tr. Dis. Bull. Vol. 4, p. 202

(196) Arch. f. Schiff's- & Tropenh., 1913, No. 17.

(197) Paper on Wild Rodents.

The Gasseni cases occurred in a locality situated on the slopes of the Kilimanjaro during the rainy season, when the natives sheltered themselves from the cold in their huts and when all kinds of respiratory diseases were frequent.

From 1915 until now the septicemic type seems to have prevailed with a corresponding decrease of the pneumonic<sup>(198)</sup>. The same condition is also found in both *Rhodesia* and *Nyasaland* where the pest has again obtained a foothold since the world war<sup>(199)</sup>.

In *Mozambique* some suspicious cases of pneumonic plague were reported in January 1899<sup>(200)</sup>.

The climate of the Uganda Protectorate, though not suitable for Europeans, is pleasant. Readings, taken in 1921 at Entebbe, show<sup>(201)</sup>:

Average rain fall for the last 22 yrs. 58.26 in.

,, temperat ,, ,, ,, 18 ,, 78.46 F. max., 62.76 min..

,, daily sunshine ..... 6 hrs. 34 min.

## 7. SOUTH AFRICA.

In South Africa an evolution of plague similar to that in the U. S. is observed. Rats became infected early in the present pandemic and outbreaks in the early years could be traced to them; only once was a species of wild rodents (*Arvicanthus pumilio*) involved. Later on outbreaks appeared which were not rat borne. Persistent research led to their connection with wild rodents, but it is not yet established how and when the latter were originally infected<sup>(202)</sup>.

Pneumonic cases were conspicuous in both series of outbreaks. Simpson<sup>(203)</sup> mentions that in Cape Town (Feb. 1901-Jan. 2, 1902; 745 cases with 362 deaths, i.e. 48.5% mortality)<sup>(204)</sup> "the pneumonic types formed 7% of the admissions and furnished a mortality rate of 70%." We may take it that all cases with lung symptoms are included in this group.

Two nurses died at this time of pneumonic plague<sup>(205)</sup>.

In 1904 (March-July) an epidemic occurred in Johannesburg, as follows:

(198) East Afr. Protect. Rep. 1915; Uganda Rep. 1916, 1920.

(199) Med. Hist. of the War. Hyg., Vol. II, p. 461; Kinghorn, qu. Tr: Dis. Bull. Vol. 13, p. 141.

(200) L. G. B. R. 1898-01, p. 175.

(201) Lancet, 1923, April, p. 730.

(202) Paper on Wild Rodents.

(203) L. c., p. 170.

(204) L. G. B. R. 1901-02, p. 325.

(205) Ibid.



In the city .....	112	with	82	deaths.
In the vicinity .....	37	,,	15	,,
<hr/>				
Total .....	149	with	97	deaths.

An epizootic among the rats was present. Pakes<sup>(206)</sup> believes the first pneumonic cases to be unconnected with any rat epizootic. It is possible that these had occurred earlier in 1904 among the coolies without being recognised by the authorities.

Dr. Gilleson (of Hankow) wrote that his brother-in-law, Dr. Marie, attended at that time an unsuspected pneumonic case and became infected. The disease spread in his family, killing all but the youngest daughter, aet. 11.

About 65 cases occurred within the first eight days in the coolie location; they were all of the pneumonic type. Then the rat epizootic became manifest and led to bubonic cases in different places.

The rapid decline of the pneumonic outbreak was certainly due to the adequate measures taken. The insanitary coolie location was evacuated (ca. 3100 persons) and burnt. The evacuated were confined in a camp for 12 days, after which they were allowed to go out, but had to live in the camp. Only one fatal case occurred in the camp within 24 days.

The pneumonic form was again seen in April 1907, when 25 cases with 14 deaths occurred in King William's Town and neighbouring localities. Rats and mice were found infected with plague as well as several cats and a pet monkey kept in a forage store<sup>(207)</sup>.

This is the last outbreak with pneumonic cases, due to rat epizootics. A few words may be added about the rats and their fleas. According to Graham<sup>(208)</sup> the predominant rat species in Cape Town is *Mus decumanus*, largely outnumbering *Cricetomys gambianus*. Rothschild reports for Cape Town *Ceratophyllus fasciatus* on *Mus rattus*; for Pretoria mainly *P. cheopis*<sup>(209)</sup>.

With regard to wild rodents the first outbreak on record (Tarka and neighbouring districts, Cape Prov.), due to the gerbille and multimammate mouse, occurred in 1914 (July-October) and claimed 35 cases with 31 deaths. It was mainly

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(206) Pakes, Rep. upon the Outbr. of Pl. on the Witwatersrand, March 18-July 31, 1904. Johannesburg, 1905; L.G.B.R. 1904-05, p. 277.

(207) L.G.B.R., 1907-08, pp. 240-241.

(208) Simpson's Gold Coast Rep., p. 25.

(209) Jl. Hyg., Vol. VI. Pl. Suppl., p. 484.

pneumonic in type. The later ones seem to have been more often of the bubonic type. It is interesting to note that B.P. was found frequently in the sputum of both patients and convalescents<sup>(210)</sup>. This matter is worth investigating.

### 8. MAURITIUS.

A survey of plague in this island appears in another article of this volume<sup>(211)</sup>.

It is probable that some pneumonic cases were present in the 1864-70 epidemic. Bruce Low mentions in his study "influenza-like symptoms.....severe bronchitis in some cases .....hemorrhages, especially epistaxis."

Pulmonary plague was certainly met with in 1899, when the existence of plague was established. The same doubt exists here as elsewhere as to the authenticity of the earlier cases, including such as are classed under 'bronchitis' but the proved cases of pneumonic plague (including that of a doctor) in May 1899 should establish the presence of this type in the early days. According to the figures submitted by Lorans there were 182 cases with 163 deaths where no buboes were seen<sup>(212)</sup>.

In almost all the later records obtainable we notice a considerable percentage of pneumonic cases among the total. This was particularly marked in 1913<sup>(213)</sup>.

In Mauritius hot weather reigns between December and April, the rest of the year being comparatively cool. The wettest month is March, the driest September<sup>(214)</sup>. The incidence of plague is highest from September to December.

Besides rats and mice, plague was found in 'musk-rats' and these insectivora are included in the anti-rat campaign. Cats were found with plague in some instances.

### 9. MADAGASCAR.

No positive records are obtainable regarding the pneumonic type in Madagascar up to 1921.

In that year a bubonic outbreak was first observed at Tamatave (February-April) traceable to a previous rat

(210) Mitchell, JI. Hyg., 1921, Dec., pp. 377-378; Rees and Targett-Adams, *qu. fr. Trop. Dis. Bull.*, Vol. 9, pp. 478-479.

(211) Orig. Home of Bubon. Pl.

(212) L.G.B.R. 1898-01, p. 184; *ibid.* 1899-00, p. 334.

(213) Denman, B. M. JI. 1914, June, p. 1236.

(214) L.G.B.R. 1901-02, p. 328.



epizootic<sup>(215)</sup>. Directly connected with this was a pneumonic invasion at Tananarive<sup>(216)</sup>, imported probably from Tamatave by one individual in the incubating stage. There was no trace of a preceding epizootic. The epidemic started from two families at a wedding in June, and lasted till end of July, claiming 46 fatal cases. Tananarive is situated on a high plateau while the outbreak occurred during the coldest season of the year.

Plague reappeared in the same locality in November 1921, lasting till Feb. 1922, i.e., during the rainy season, when the rats migrate from the rice plantations in the nearest houses<sup>(217)</sup>. There were:—

Bubonic c. ....	31 (4 with sec. pneum.)
septic. ....	15
pr. pneum. ....	2
<hr/>	
Total .....	48

The U.S.P.H. Rep. for Tananarive (Jan. 1-Dec. 10, 1922) are:—

bubon. ....	37
septic. ....	28
pneumon. ....	8 (10.9%)
<hr/>	
Total .....	73

Pneumonic cases were reported also from other parts of the island, especially in the Moramanga province and are still present<sup>(218)</sup>.

## VII. PNEUMONIC PLAGUE IN WESTERN EUROPE.

### 1. PORTUGAL.

Reports of the *Oporto* epidemic (1899) are again accompanied by a notable increase in deaths from 'respiratory diseases and tuberculosis'. Positive pneumonic cases were encountered with bubonic ones in the autumn of 1899<sup>(219)</sup>.

(215) Goyon, Bull. Soc. Path. Ex., 1921, pp. 602-609.

(216) Gouzien, *ibid.*, pp. 610-621; Bouffard and Girard, *ibid.*, 1923, pp. 501-523; Allain, Ann. de Med. and Pharm. Colon., 1921, pp. 379-418.

(217) Bouffard and Girard, *l. c.*

(218) U.S.P.H.R., 1923, June 29, p. 1509.

(219) L.G.B.R. 1898-01, p. 67; Calmette, Rev. d'Hyg., 1899, 11; Zabolotny, Pestis Bubonica, 1907, pp. 82-84; Frosch and Kossel, Klin. Jahrb., VII. p. 473.

Professor Camara Pestana, while performing a *post mortem* upon a case of *pneumonic* plague on November 7, received a small scratch on his left middle finger. He fell sick after his return to Lisbon on the 10th, developing first a left axillary and supraclavicular bubo and then secondary lung symptoms. He died on the 15th.

The existence in Oporto of "some cases of infectious pneumonia" was officially admitted in 1904, but no particulars were given<sup>(220)</sup>.

Faria<sup>(221)</sup>, giving the records of plague in *Lisbon* to be *pneumonic* 8 out of 10 seen in October 1914.

Sousa<sup>(222)</sup> saw a number of *pneumonic* and *septicemic* cases in a mild outbreak in *Azores* (1908).

## 2. FRANCE.

A few suspicious cases of fatal pneumonia, preceding a small plague outbreak, occurred at Marseilles in September 1903<sup>(223)</sup>.

## 3. ENGLAND AND SCOTLAND.

Two groups of outbreaks should be distinguished: one directly due to ship importation and caused by rats in ports, the other developing slowly among *M. decumanus* found under rural conditions and among free living rodents (rabbits, hares) which had become secondarily infected through ship rats.

The *pneumonic* variety was inconspicuous in the first group, but conspicuous in the second.

Some *pneumonic* cases undoubtedly occurred during the Glasgow epidemic of 1900<sup>(224)</sup> especially at beginning<sup>(225)</sup>. Further records of definite cases are absent<sup>(226)</sup>.

In our paper on Wild Rodents mention was made only of the role of the rabbits and hares. A few words about the important rats may now be added. *M. decumanus*, practically the only rat present in the affected districts in East Suffolk, was found<sup>(227)</sup> to lead a rural life in summer, while in winter it dwelt near the farm houses. It was found infested with

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(220) L.G.B.R. 1904-05, p. 272.

(221) Qu. fr. Tr. Dis. Bull., Vol. 19, p. 727.

(222) L.G.B.R. 1908-09, p. 25.

(223) Ibid., 1903-04, p. 276.

(224) Zabolotny, Mukden Rep., p. 159.

(225) L.G.B.R. 1898-01, p. 57.

(226) L.G.B.R. 1901-02, pp. 309-310.

(227) Martin and Rowland, Observ. on Rat Pl. in East Suffolk.



*Ceratophyllus fasciatus* and *Ctenophthalmus agyrtes*, the first attacking man readily, the latter never in 70 experiments carried out. Plague infection among these rats was probably caused by their fellows escaping from grain ships bound for Ipswich<sup>(228)</sup>. It had existed presumably since 1906 or even earlier<sup>(229)</sup>. The rabbits and hares were apparently infected from the rural rats, which occasionally used the rabbit holes. Besides *Spinopsyllus cuniculi*, *Ceratophyllus* was occasionally found on the rabbits<sup>(227)</sup>.

The human outbreaks in this area are herewith tabulated:

1906-07	Shotley	.....	8 c. with 6 d. (75%)
1909-10	Trimley	.....	8 c. ,, 5 d. (62.5%)
1910	Freston	.....	4 c. ,, 4 d. (100%)
1911	Shotley	.....	1 c. ,, 1 d. ,,
1918	Erwarton	.....	2 c. ,, 2 d. ,,

The first two outbreaks were not recognised as plague at the time but later investigation made this diagnosis clear. In Shotley (Dec. 1906-Jan. 1907) 3 males (aet. 56, 7, ?) and 5 females (aet. 53, 46, 24, 19, ?) took ill; two recovered. In Trimley (Dec. 1909-Jan. 1910) 3 males (aet. 50, 12, 6) and 5 females (aet. 46, 18, 14, 9, 7) got sick, of whom five died. Poisoning was at first suspected and three inquests and two *post mortems* were performed. The type of plague met with was not definitely stated in those two outbreaks<sup>(230)</sup>; possibly the pneumonic form played some part<sup>(231)</sup>.

The visitation at Freston (Sept. 1910) was pneumonic. Bulstrode<sup>(232)</sup> thinks, however, that the lung symptoms in the first victim, (girl aet. 9) were secondary. This was followed by the mother (aet. 40), the father (aet. 57) and a woman (aet. 43). Three other children of the family (aet. 14, 13, 7) and 4 grown-up contacts remained healthy.

The seaman, who fell sick in Shotley in 1911, was apparently infected by one or the other of two wild rabbits with which he had been in contact on October 7. He fell sick 3 days afterwards, with axillary bubo and secondary pneumonia and died<sup>(233)</sup>.

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(228) L.G.B.R. 1914-17, p. 146.

(229) Lancet, Dec., 1911, p. 1835.

(230) Ibid.

(231) Osler, Princ. and Pract. of Med., 1918, p. 138.

(232) L.G.B.R. 1910-11, p. 36; Brown and Sleigh, B. M. J., Nov. 12, 1910.

(233) Browne, qu. fr. Trop. Dis. Bull. Vol. I, pp. 549-550; L.G.B.R. 1912-13, p. 65.

In June 1918 two fatal cases of pneumonic plague were reported<sup>(234)</sup> from Erwarton, Samford Rural District, of two women living in two neighbouring houses in the country. The second victim was infected when nursing the first.

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## VIII. PNEUMONIC PLAGUE IN EASTERN EUROPE AND THE NEAR EAST.

### 1. GREECE.

One pneumonic outbreak in Syros, Cyclades Islands, is mentioned in the L.G.B.R.<sup>(235)</sup> and described apparently by Kuriazides<sup>(236)</sup>. This epidemic, taking place in January 1916 and connected with grain, imported from the Piraeus, was seemingly due to a mixed infection of B.P. and diplococci. Though dead rats had been seen before the outbreak, it was not possible to prove infection in the rodents captured. There were 20 cases with 14 deaths.

### 2. TURKEY IN EUROPE.

Only two doubtful references could be found. One concerns a "serious and fatal epidemic of pneumonia" raging in Yannina (Epirus) early in 1901<sup>(237)</sup> the other a confirmed plague case, occurring about the same time and displaying pneumonic features<sup>(238)</sup>.

### 3. ASIATIC TURKEY.

Plague in its pneumonic form was observed in December 1900 near *Smyrna*. This city itself had been visited by purely bubonic plague in May and June. The pneumonic outbreak was seen in the adjacent village of Thomaso, imported by a sick man from Smyrna. Up to January 2, 13 cases with 12 deaths were counted in three families, almost all the attacked suffering from pneumonic plague. The diagnosis was not confirmed bacteriologically, but inasmuch as the disease had been preceded by true plague cases in Smyrna there seems no reason to be sceptical<sup>(239)</sup>.

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(234) L.G.B.R. 1918-19, p. 82.

(235) 1914-17, p. 134.

(236) Qu. fr. Trop. Dis. Bull., Vol. 9, p. 86.

(237) L.G.B.R. 1898-01, p. 129.

(238) Ibid., p. 127.

(239) Ibid., pp. 129-132; Loutfi Bey and Mizzi, La peste a Smyrne en 1900, Constantinople, 1900.



It was reported<sup>(240)</sup> that the cases occurring singly or in groups in *Adalia* (Asia Minor) annually since 1905, were often of the pneumonic type. The infection was sometimes imported, sometimes a local rat epizootic was noted. Plague occurred most frequently in summer.

#### 4. SYRIA.

Russell<sup>(241)</sup> studying the epidemic at *Aleppo* in 1760-62 found no lung symptoms in 2700 patients.

In *Beyrout* the pneumonic type was noted as in *Adalia*<sup>(240)</sup>. One such outbreak (1908-09) was described by De Brun<sup>(242)</sup>. Three earlier cases of unknown origin had been observed in the town in September 1908<sup>(243)</sup>. De Brun states that an old man died in the town about the middle of December and 'pneumonia' was believed to be the cause of death. His wife, falling sick, was admitted into the French Hospital on the 19th and died on the 20th. She showed no physical signs of pneumonia, only dyspnoea. Her cough was dry almost up to the end, when bloody frothy sputum was noted (oedema?). The Sister of Mercy, who attended her, also fell sick on the 24th and died after three days, followed by three other sisters who had slept in the same room. All had pneumonic symptoms.

#### 5. ARABIA.

Xanthopoulides maintains that in the *Djeddah* outbreak from February to June 1899 only bubonic cases were registered, while fatal cases of pneumonia, occurring at the same time, were not considered as plague<sup>(244)</sup>. He further asserts that the numerous cases of slight illness without buboes, but with some respiratory symptoms were caused by B.P.; he saw in such cases bacilli similar to B.P. No further tests were made, to identify them<sup>(245)</sup>. No epizootic was noted at the time of the epidemic; this had been confirmed during previous outbreaks and a wholesale destruction of rats had been carried out<sup>(245)</sup>.

Pneumonic cases seem to have been present in *Djeddah* during a small outbreak in 1913 (32 cases with 28 deaths). It was stated<sup>(246)</sup> that this epidemic did not take place during the usual plague season (Jan. March) but appeared in March

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(240) L.G.B.R. 1909-10, p. 57.

(241) A Treat. on the Pl., London, 1791.

(242) Bull. Acad. Med., 1909, p. 61.

(243) L.G.B.R. 1908-09, p. 31.

(244) Rapp. sur la recente manifest. de peste à Djeddah, 1899.

(245) L.G.B.R. 1898-01, pp. 149-151.

(246) Ibid., 1913, p. 62.

and lasted till June. One of the last sufferers was the registrar of the British Consulate who showed undoubted pneumonic symptoms and died after a short illness.

The high mortality of an earlier (1910) outbreak with 98 deaths among 100 cases seemed to point to pulmonary plague<sup>(247)</sup>. It is possible that mild cases remained unnoticed. Dr. Zonchello, the acting sanitary officer, who had performed necropsies and bacteriological examinations, died from the pest.

Plague in the old endemic area of *Assyr* seems mainly bubonic. Our scanty epidemiological knowledge of this area must be kept in mind.

#### 6. MESOPOTAMIA.

During the outbreaks about 1880-90 pneumonic symptoms were observed; they were apparently secondary to bubonic plague<sup>(248)</sup>.

In the present pandemic the pneumonic type seems rare. Pulmonary symptoms and bloody sputum were seen by the Turkish military surgeons who had been sent out to investigate an outbreak in Zobeir on the right bank of the Tigris (April 1903)<sup>(249)</sup>.

During the world war, when this area received better study, only the bubonic type was reported. One pneumonic case occurred in an Indian sepoy<sup>(250)</sup>.

#### 7. PERSIA.

The bubonic type is found in those parts of Persia belonging to the Mesopotamia endemic area; occasional pulmonary cases were noted<sup>(251)</sup> but never a pneumonic outbreak. The part played by the common rats and their fleas (mainly *X. cheopis*, rarely *Ceratophyllus fasciatus*) has now been established in this region<sup>(250)</sup>.

The same does not hold regarding epizootics and epidemics in the province of *Khorassan*. A Russian observer, Grekoff, found infection in the *field* rodents, not the rats, and the single human outbreak recorded in detail was pneumonic (Turbat-i-Sharkh-Djami, October 1912), as clinically diagnosed by Sorotin. 50 death occurred<sup>(252)</sup>.

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(247) Ibid., 1910-11. p. 165

(248) Sticker, l. c. Vol. I. pp. 333 and 339.

(249) L.G.B.R. 1903-04. p. 281.

(250) Mod. Hist. of the War, Hyg., Vol. II. p. 471.

(251) Clemow, Lancet, 1913. Vol. II, p. 1576.

(252) Clemow, Lancet, 1912. Nov., p. 1323; Egypt. Pl. Rep., 1923, p. 45.



## 8. SOUTH RUSSIA.

- A. Some of the 83 patients affected with plague in Tutshkoff (on the Danube) in winter 1824-25 showed pneumonic signs<sup>(253)</sup>.
- B. During the plague outbreak of 1910 at Odessa some cases of primary pneumonia were recorded by Chenzinski<sup>(254)</sup>. The viscera of one patient had a curious story: A man in Petrograd found in the street a box, containing jars with human viscera. He deposited this at the Police station, where the contents were claimed by the doctor who had brought them from Odessa<sup>(255)</sup>.
- It would appear that sporadic pulmonary cases had occurred in Odessa during previous outbreaks<sup>(256)</sup>.
- C. An outbreak of 'infectious pneumonia' was reported in the Caucasus (village Tchiatoury) in May 1900. Plague was suspected although a laboratory expert came to negative conclusions<sup>(257)</sup>.

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IX. PLAGUE IN ASTRAKHAN AND ADJOINING TERRITORIES.

These endemic areas are so important in the history of pneumonic plague that it may be useful to give also some data regarding non-pulmonary outbreaks.

## 1. ASTRAKHAN.

No definite records exist up to the time of the Black Death. The chronicler Nestor speaks of outbreaks of 'pestilence' in A. D. 1090 and 1092, but it is not clear if these were plague. The same may be said of invasions in the 12th and 13th centuries<sup>(258)</sup>.

Zabolotny<sup>(259)</sup> gives the following data:—

1364 Volga low-lands (& Central Russia)  
1374-77 Whole of Russia

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(253) Sticker, l. c., p. 295.

(254) Plague in Odessa, 1910.

(255) L.G.B.R. 1910-11, p. 167.

(256) Sticker, l. c., p. 380; Marchand, Muench. M. W., 1903, pp. 1658-1659.

(257) L.G.B.R. 1898-01, p. 124.

(258) Papers on Pl., edit. by the Russian Pl. Comm., Part II, Petrograd 1907.

(259) Pestis bub., Petrograd, 1907.

1654-57	Volga low-lands (& other parts)
1692	Astrakhan
1727-28	„
1806-08	„ (& Saratov)

The Official Report<sup>(258)</sup> mentions others in the 15th and 16th centuries.

Russian authors generally state that these outbreaks were due to importation from the *west*. One exception was the terrible outbreak in Astrakhan from July 1692 to December 1693, which killed over 10,000 out of 16,000 inhabitants. The origin of that epidemic is traced back to the eastern foci of plague. It is worth remembering in this connection that about 1630 "a new route to Persia through the Caspian Sea, Astrakhan, Novgorod and Narva was.....opened, by which for a considerable time the produce of Persia and the East was conveyed to France"<sup>(260)</sup>.

Derbek<sup>(261)</sup> assumes that the 1634 outbreak started from the Volga low-lands and that the later 1727-28 epidemic came from Persia.

The 1806-08 epidemic, claiming 1809 victims in the district and 650 in the city of Astrakhan was believed to arise from the Caucasus. The *Vetlianka* outbreak (1878-79) which rivetted attention upon this endemic area, was preceded in 1877 by bubonic cases in other localities as well as in the city of Astrakhan. There exists a controversy as to the origin of these epidemics of *pestis minor*. Some consider it to be Asia Minor, from which the Russian army returned after the Turkish war, but the majority of Russians suspect Persia, where in 1877 a great epidemic killed a tenth of the inhabitants of Resht. Later observers<sup>(262)</sup> maintain that plague might have been endemic in Astrakhan at the time already.

While it is possible that the *Vetlianka* epidemic was a continuation of the *pestis minor* (some say that the first case came from Astrakhan)<sup>(263)</sup> others believe that it was directly imported from the theatre of war<sup>(259)</sup>.

This lasted from Oct. 1878 to May 1879, claiming

In Vetlianka .....	454 c. with 372 d. (81.9%)
In 7 neighb. vill. ....	66 c. with 62 d. (93.9%)

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Total .....	520 c. with 434 d. (83.4%)
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(260) Simpson, p. 33.

(261) History of Pl. Epid. in Russia, Breslau, 1906.

(262) Klodnitzki, Astr. Conf. Rep. (1910). p. 100.

(263) Silberberg, *ibid.*, p. 91.



It appears that up to November the disease was purely bubonic; Hirsch states<sup>(264)</sup> that the majority of cases were *pestis minor*.

From November onwards pneumonic cases appeared frequently. Muench observed that pulmonary cases arose in families stricken by bubonic plague and that contacts of pneumonic patients developed bubonic plague. Thus he clearly recognised both forms to be due to one and the same infection, while the earlier observers classed the lung cases under 'pneumotyphus' and other vague names. Muench found lung symptoms in 62 cases; the Official Report adds 26 more misdiagnosed as 'pneumo-typhus' etc., so that there was a minimum of 88 pneumonic cases, occurring mainly at the height of the epidemic (Nov.-Dec.-Jan.) From February onwards the cases were again mainly bubonic.

The mortality among the *sanitary staff* was high:

Doctors	.....	3	(pneumon. 3)
Feldchers	.....	7	„ 1
Others	.....	14	„ 1 (Sister)
<hr/>			
Total	.....	24	(pneumon. 5)

One priest also died. The Russian Report adds that the high death-rate among the staff was due to their disbelief in the plague nature of the outbreak. Muench found only 55.6% morbidity among families of the sick.

Payne (one of the members of the British Commission sent into Vetlianka) stated<sup>(265)</sup> that "a large mortality among rodents was observed, but its relationship to the epidemic of plague did not impress him at the time. Now he is inclined to think, that the association was very intimate."

Two further outbreaks were reported in 1880-1890:

- A. One with 40 victims related by a layman in a village of the Krasnojarsk district with bubonic features<sup>(266)</sup>.
- B. One on an island in the Caspian Sea (near Dshambaja) among the Kirghese. A steamer was sent there, but none of the crew dared go ashore and threw two bottles of brandy instead. All the inhabitants died. The nature of this outbreak is doubtful, because cerebral fits were the principal symptoms<sup>(266)</sup>.

(264) Hirsch and Sommerbrodt, Mitt. ueb. d. Pestep. i. 1878-79 in A. Berlin 1880.

(265) Simpson, p. 99.

(266) Off. Rep., pp. 144-145.

A long clear interval followed. The pest might exist but was not reported<sup>(267)</sup>.

1896. *Aksai. Mainly bubonic.* The local doctor observed in autumn 1896 an epidemic (60 c. with 42 d.) in 17 houses; 3 families died out. Though buboes (mainly inguinal) were present, he diagnosed 'typhus.' Isolation measures adopted by the inhabitants prevented a further spread of the disease.

This outbreak remained unnoticed until 1902, when a plague commission visited the spot and was satisfied that the 1896 malady was plague. Some of the cases were found to be pneumonic<sup>(268)</sup>.

1899. *Koloborka. Mainly pneumonic*<sup>(269)</sup>. (24 c. with 23 d.) Before the middle of July 1899 there had been no deaths in the village suggesting plague, as could be seen from the church books. The first case occurred in a woman, aet. 37, who fell sick in the fields on July 16 and died at her house after 5 days. She had bloody sputum and the Russians<sup>(270)</sup> consider her case as *pneumonic* plague. It must be noted, however, that this woman and other patients after her were not examined by doctors.

All the later cases could be traced to this woman. They were mainly of the pneumonic type, although the outbreak, lasting up to August 9, occurred during the summer. Some septicemic cases were also present; in one the p.m. showed no pneumonic foci, only hyperemia in the lungs. Five cases, including the surviving one, appearing at the end, had buboes (inguinal, axillary, cervical) and often secondary lung involvement. Large hemorrhages in the skin were observed in some.

1899. 3 *Islands in the Caspian Sea. Mainly pneumonic*<sup>(271)</sup>. These islands were inhabited by Kirghese, who dwelt in poorly built and dirty houses, abounding with vermin, especially fleas. *Kishkene-aral* and *Kene-aral* were separated from each other by a narrow channel, while *Irsali-aral* was 24 E. miles distant.

(267) Off. Rep., p. 161; Konstanoff, Vj. Guig., 1912, Febr., pp. 178-194; Klodnitzki, l. c.

(268) Klodnitzki, R. Vrach, 1913, No. 30-31.

(269) Levin, Vrach, 1899; Arustamov, Schmidt, Vrach, 1900; Marmelstein, Weekly f. Pract. Med., 1900, p. 82; Chausov, Jl. Mil. Med., Jan. 1900; Tartakovski, Vrach 1900, No. 33.

(270) Berestneff, R. Vrach, 1906, pp. 789-792.

(271) Arustamov, Vrach, 1900, p. 457.



A. *Irsali-Aral* (52 inhab. in 18 huts; 10 huts affected).

<i>Age and sex:</i>	<i>Tot. popul.</i>	<i>Sick:</i>	<i>Died:</i>	<i>Record.</i>	<i>Healthy</i>
Adult m. ....	13	13	11	2	0
„ f. ....	15	7	7	0	8
11-15 yrs. ....	9	4	3	1	5
Children ....	15	1	1	0	14
	<hr/>	<hr/>	<hr/>	<hr/>	<hr/>
Total .....	52	25	22	3	27

This outbreak lasted from October to December and was mainly pneumonic, though a few bubonic cases were present. All three cases of recovery were reported to have fever and cough but no buboes.

B. *Kishkene and Kene-arals* (64 inhabitants in 16 huts).

<i>Age and sex:</i>	<i>Tot. popul.</i>	<i>Sick:</i>	<i>Died:</i>	<i>Record.</i>	<i>Healthy</i>
Adult m. ....	15	12	12	0	3
„ f. ....	23	15	15	0	8
Children ....	26	12	12	0	14
	<hr/>	<hr/>	<hr/>	<hr/>	<hr/>
Total .....	64	39	39	0	25

The age incidence of the 12 affected children:

3 months—1; 2 yrs.—2; 4 yrs.—3; 5 yrs.—1; 8 yrs.—2; unknown—3.

Cases appeared simultaneously in both islands towards the middle of November. The panic stricken tried to leave Kishkene-aral but a raging storm prevented them. They managed to reach the other island where the doctors found in one hut 5 dead bodies, the remnants of three families. In another hut 12 persons belonging to 2 families had taken shelter and the doctors found 11 dead with one dying girl. Some cases showed buboes, but the pneumonic type prevailed.

1900. *Vladimirovka. Bubonic*<sup>(272)</sup>.

This village (8-9000 inhab.) lies 47 E. miles from Kolo-bovka. The outbreak started in the fields, a feature characteristic in Astrakhan where infection originates in the wild rodents. It lasted from Nov. 4 to Dec. 17.

<i>Age and sex:</i>	<i>No. contacts:</i>	<i>Sick:</i>	<i>Died:</i>	<i>Record.</i>	<i>Mild:</i>	<i>Healthy.</i>
Adult m. ....	8	5	5	0	1	2
„ f. ....	16	9	7	2	1	6
Adolescent .....	3	1	1	0	1	1
Children .....	8	3	3	0	0	5
	<hr/>	<hr/>	<hr/>	<hr/>	<hr/>	<hr/>
Total .....	35	18	16	2	3	14

(272) Fedorov, Rep. Astr. Med. Soc., 1909, No. 1; Messarosh, Vj. Guig., 1901, p. 991.

Distribution of buboes in 18 cases:

Inguinal—11; axillary—2; cervical—1; no bubo—1; undeterm.—3.

In three of the six p.m. the lungs were found secondarily affected.

1900-01. *Tekebai-Tubek*. *Pneumonic*<sup>(273)</sup>.

This epidemic is noteworthy for several reasons. It began at the end of Nov. 1900 at *Atshikbai*, where a Kirghese, aet. 50, fell sick. The infection of other localities: *Tekebai-Tubek* (14 E. miles), *Mereke* (6 miles) and *Karakuga* (53 miles) can be traced to *Atshikbai*. The epidemic lasted until Jan. 11 and claimed 151 deaths with 14 alleged recoveries.

a. Affected huts (in 3 localities):

In 15 with 74 inhabit. died 74 (all)

In 6 „ 52 „ „ 46 (1 rem. in each)

In 7 „ 48 „ „ 19 (end of epidem.)

b. Age incidence (in 3 local.):

Age:	Total:	Died:	%
Up to 10 yrs. ....	54	37	68.5
11-20 yrs. ....	23	20	86.9
21-40 „ ....	55	46	83.6
41-60 „ ....	22	22	100.0
Over 61 yrs. ....	20	14	70.0
Total .....	174	139	79.8%

c. Age and sex of sick (Konstanzoff):

Age:	71-90.	51-70.	31-50.	21-30.	11-20.	3-10.	Younger.
Died male .....	0	10	17	23	12	34	13
Died female .....	1	7	10	16	8		
Total .....	1	17	27	39	20	34	13

N.B. Konstanzoff thinks that the most exposed were 26-50 yrs.

Dr. Gos who first reached the affected area at the end of December, wrote: Sick and healthy persons were found surrounded by corpses; they were often unable to get food or to heat the huts. In other instances the sufferers were driven out into the cold by their frightened families. The inhabitants of the healthy compounds did not dare to go near

(273) Off. Rep., pp. 67-99; Konstanzoff, Vj. Guig., 1902, pp. 1491-1515.



to the affected huts; they watched whether smoke was still coming out of the chimney or if there was anybody left to light a fire. The cattle was gathered round the huts where all had died, asking for food.

Other features deserve special mention :

- i. The outbreak, so far as it was seen by the doctors, was purely of the pneumonic type, but the first cases were not examined.
- ii. Some cases were due to a mixed infection. This was never diagnosed clinically, but of the 8 dead bodies which were thoroughly investigated (altogether 14 p.m. were performed), 2 only presented a pure infection with B.P., while 4 showed also pneumococci, 2 streptococci, one of them being a woman who died 2 days after child birth. It was—as the observers aptly point out—due to this mixed infection that a lobar type of plague pneumonia (without affection of the bronchial glands) was mainly met with. The virulence of the plague strains, when tested afterwards in the laboratory, was found to be moderate (Konstanzoff) and it was stated (Off. Rep.) that this lessened virulence was conspicuous at the end of the epidemic.
- iii. The alleged recoveries are hard to explain. They read like the stories of the 1910-11 Manchurian epidemic. Only in one case were suspicious bacilli seen in the sputum. The other cases, including Dr. Gos's, who attended a sick feldcher (dresser), are not proved.
- iv. Both serum and Haffkine's vaccine were administered prophylactically, the former being given to the most exposed. Among the 43 inoculated persons taken out of the infected huts, no case resulted, while 19 were seen among the uninoculated.
- v. The inhabitants were evacuated from the infected houses into *kibitkas* (canvas-covered wagons). 46 persons were removed. One group who stayed were healthy throughout, while another of 16 returned after a few days to their house where two fatal cases had previously occurred. Soon afterwards a woman (aet. 27) and a child (aet. 4) died and another a week after. As the incubation period in this outbreak was generally 2-4 days, it is likely that the house was infectious.

- vi. It is possible that some of the children did not succumb to plague infection but to want of care.
- vii. At the end of the epidemic a medical inspection of the inhabitants in the Kirghiz steppes showed no plague cases among 380,000 persons, but in an adjacent district over 100 persons displayed scars in the inguinal region due probably to an attack of mild plague. Most of them had been sick in 1898 or 1899.

1902. *Aksai. Mainly bubonic*<sup>(274)</sup>. This outbreak, lasting from end of May till July 27 and causing 35 cases with 20 deaths, was bubonic with the exception of 2 pulmonary cases and one of plague meningitis.

The buboes were distributed as follows:

Inguinal—14; femoral—5; axillary—7; round ear—4; cervical—2; ? front chest—3; total 35 (including some doubtful cases).

The fatal ones had skin hemorrhages, sometimes extensive.

1902. *Ush-Kuduk. Limited bubonic.*

A small outbreak happened in June (6 cases with 5 d., including one pneum.)<sup>(275)</sup>. Previous to this a more widespread one near Tekebai-Tubek (Sept. 1901-Apr. 1902) consisted of *pestis minor*. The distribution of the buboes was as follows:—

Cervical—18; inguinal—11; axillary—6; auricular—3; total 38<sup>(276)</sup>.

1903. *Bikovo. Mainly bubonic.* Bikovo, a big settlement, lies further northwards on the Volga, 360 miles from Astrakhan City. The outbreak lasted from August till November. Anthrax cases were present at the same time. Though no cases of mixed infection were seen, the Official Report suggests the possibility of B. Anthr. overgrowing the B. P.

The records<sup>(277)</sup> (including some doubtful cases) showed:—

Cervic. buboes—3 with 2 d.; axillary—3 with 2 d.; inguinal—3 with 3 d.; pneumonic pl.—3 with 3 d.; anthrax—5 with 2 d.

(274) Gos, Vj. Guig., 1903, p. 337; Schmidt, Pl. in Aksai, 1902.

(275) Konstanoff, *ibid.*, 1903, pp. 1282-1307.

(276) Weinstein, *ibid.*, 1902, p. 1451.

(277) Vinogradoff, *ibid.*, 1904, p. 1136.



1904. *Kos-Chagil. Imported pneumonic.* A family was wiped out in December; when the doctors examined the corpses, buboes (inguinal and submaxillary) could be noted on two children; the other 5 had pneumonia. The infection was imported from the Ural Province, where plague raged<sup>(278)</sup>. Deminski<sup>(279)</sup> emphasizes that this is the only modern example of imported plague into the endemic areas of Astrakhan.

1905-06. *Beketaer plague. Pneumonic*<sup>(280)</sup>. Scanty information about this is available. It spread over a large district of 42 localities and claimed 1000 cases. The exact origin is unknown; it started presumably in summer and by October patients with cervical buboes were seen. Later on, the type was pneumonic with few bubonic cases. The epidemic lasted till March, reaching its height in October and November.

No research work could be carried out, because the energy of the medical staff was absorbed in fighting the pest. The nature of the houses (see our concluding remarks), the cold, scarcity and bad quality of the water made disinfection difficult. It was even asserted that new cases started, after the houses had been disinfected; so the huts were burned<sup>(281)</sup>. This statement was much criticised by other observers<sup>(282)</sup>, who claimed that only two or three such instances occurred, and no deductions should be drawn.

The natives suspected that camels were the source of the outbreak, while an observant layman<sup>(283)</sup> said he saw corpses of wild mice round the pest huts.

1906. *Usag-Bai. Bubonic.* One family died from plague in Usag-Bai (April-May) supposed to come from a pillow and mattress taken from an infected locality<sup>(284)</sup>. Two other cases occurred at the same time in *Altai* (50 miles distant)<sup>(285)</sup>.

1907. *Astrakhan City. Doubtful.* One illness, affecting three members of a family living far from the endemic area, was possibly caused by a plague-like organism (*B. lactis aerogenes* group)<sup>(286)</sup>. The *post mortem* findings were unlike plague.

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(278) Filaretov, *ibid.*, 1905, p. 683.

(279) *Ibid.*, 1912, p. 1329.

(280) Berestneff, R. Vrach, 1906, pp. 789-792.

(281) Aiklender, *Astr. Rep.*, pp. 248-249.

(282) Klodnitzki, *ibid.*, p. 282; Deminski, *ibid.*, p. 447.

(283) Rogovenko, *qu. by* Klodnitzki, *ibid.*, p. 119.

(284) Deminski, *Astr. Rep.*, p. 28, Vj. Guig., 1912, p. 1323.

(285) Deminski, *Astr. Rep.*, p. 28.

(286) Klodnitzki, *Astr. Rep.*, p. 102.

1907. *Pestchanka, Saratov Gov. Bubonic*<sup>(287)</sup>. The first patient (boy, aet. 11) had transported goods through the endemic areas. As the party avoided all settlements by staying in the open at night time, he must have been infected more or less directly by wild rodents. No hunting was done and the boy's sickness rather suggests an infection through flea-bite. He complained of having been bitten on the neck by an insect and developed a cervical bubo. Out of 24 exposed, there were:

Sick with cerv. bubo—1; with axill.—1; with inguinal—6; total 8 cases with 2 deaths.

1907. *Dshalpak-Utkul. Probable plague*. This outbreak, taking place in July in a Kirghiz camp, claimed 7 members of a family of 13 and 2 other victims who had taken part in the burials.

Only the decomposed corpses were seen by the doctors, who found B. P. and involution forms in the smears. The first case (boy, aet. 8) had some throat trouble (tonsillar plague), the last two possibly primary pneumonia; others showed buboes<sup>(288)</sup>.

Popular opinion suspected the camel as the source of this outbreak<sup>(289)</sup>.

1907 (Oct.). *Tas-Aral. Plague*<sup>(290)</sup>. Three persons fell sick after eating the meat of a camel dead of anthrax. The first two were stated to have buboes<sup>(291)</sup>, the third showed at p.m. hemorrhagic enteritis. From animals infected with material from this dead body a pure plague culture was obtained. This may be an example of the intestinal type of plague.

The head of the family who had dissected the camel, remained healthy; the role of this particular animal seems unproven.

1908. (June-Aug.). *Kul-Taban and Saraldshindikul. Mainly bubonic*<sup>(292)</sup>. The first eleven patients had buboes; 9 died. The twelfth had fatal pneumonic plague.

1909. *Beis-Kulak, etc. Mainly pneumonic*<sup>(293)</sup>. This epidemic mainly attacked Kirghiz settlements, containing poorly built houses. Many fleas and lice, but no bugs were

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(287) Galler, Vj. Guig., 1907, p. 1691.

(288) Klodnitzki, Astr. Rep., p. 105.

(289) Deminski, Vj. Guig., 1912, p. 1329.

(290) Rep. Astr. Med. Soc., 1908, Jan., p. 39.

(291) Deminski, Astr. Rep., p. 35.

(292) Bjelilovski, Rep. Astr. Med. Soc., 1908, No. 7-8.

(293) Bjelilovski, Astr. Conf. Rep., pp. 287-375.



seen in the houses. Rats were absent and no trace of any epizootic was found.

The first affected, Beis-Kulak, is situated not far from the borders of the Ural Province and Bjelilovski asserts that the infection was imported from there. The disease was imported by human agency into *Kushuk-Kirgan* (33 miles away) and probably thence into *Akbalik*. How the locality *Naur sali-Chagil* became infected, is obscure<sup>(294)</sup>. The plague was conveyed to *Bodai* and *Novoe Kasanka* by one man.

a. General survey of the epidemic:

<i>Locality:</i>	<i>Bubon.</i>	<i>? Septic.</i>	<i>Pneum.</i>	<i>Total.</i>	<i>Healthy.</i>
Beis-Kulak .....	3	4	7	14	15
Kushuk-Kirgan .....	0	0	17	17	3
Akbalik .....	0	0	28	28	13
Naur sali-Chagil .....	0	0	28	28	30
Bodai .....	0	0	20	20	31
Novoe Kasanka .....	1	0	8	9	7
Total .....	4	4	108	116	99

b. Age and sex incidence of infected:

<i>Age:</i>	<i>Male:</i>	<i>Female:</i>	<i>Total:</i>	<i>%</i>
0-1 .....	0	0	0	0.0
1-5 .....	2	6	8	6.88
5-10 .....	4	5	9	7.74
10-15 .....	3	2	5	4.30
15-20 .....	2	5	7	6.02
20-30 .....	17	6	23	19.78
30-40 .....	19	3	22	18.92
40-50 .....	15	2	17	14.62
50-60 .....	8	6	14	12.04
60-70 .....	3	5	8	6.88
70 and more .....	0	3	3	2.58
Total .....	73	43	116	

Some salient points of this well observed outbreak may be discussed:

- i. The diagnosis was confirmed experimentally and by 9 *post mortems*. In three or four of them no marked pneumonic signs were present corresponding to the 'pulmonary' cases seen by us in Harbin 1921.

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(294) Deminski, Astr. Conf. Rep., p. 33.

- ii. The mean incubation period was 4-5 days; sometimes it was reduced to a few hours, sometimes prolonged (8, 8, 9, 10 days in 4 pn. cases). Bjelilovski therefore suggests a longer quarantine than the usual 5 days.
- iii. Interesting symptoms were large skin hemorrhages in some instances and a foul-smell from the mouth. This sign—if really due to plague—would bring back the memory of the Black Death.
- iv. The length of illness was up to 5 days. Serum administration seemed to prolong it.
- v. The fact that all children under one year escaped infection, though they were in close contact with patients, seems remarkable.
- vi. In the affected districts 1339 persons (including 177 immediate contacts) received prophylactic serum (adults 40 c.c.; children 3-15 yrs.—20 c.c.; 1-3 yrs.—10 c.c.). No definite conclusions can be arrived at from the figures supplied as to its value.

*Cases from winter 1909-1912<sup>(295)</sup>:*

<i>Months</i>	<i>1909</i>			<i>1910</i>			<i>1911</i>			<i>1912</i>			<i>Total</i>		
	<i>S.</i>	<i>D.</i>	<i>R.</i>	<i>S.</i>	<i>D.</i>	<i>R.</i>	<i>S.</i>	<i>D.</i>	<i>R.</i>	<i>S.</i>	<i>D.</i>	<i>R.</i>	<i>S.</i>	<i>D.</i>	<i>R.</i>
Jan.	—	—	—	27	27	—	37	37	—	22	22	—	86	86	—
Feb.	—	—	—	30	30	—	—	—	—	2	2	—	32	32	—
March	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
April	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
May	—	—	—	—	—	—	8	8	—	—	—	—	8	8	—
June	—	—	—	—	—	—	7	7	—	—	—	—	7	7	—
July	—	—	—	13	13	—	14	14	—	—	—	—	27	27	—
Aug.	—	—	—	—	—	—	14	14	—	6	4	2	20	18	2
Sept.	—	—	—	—	—	—	22	22	—	—	—	—	22	22	—
Oct.	—	—	—	6	6	—	62	61	1	—	—	—	68	67	1
Nov.	27	27	—	64	63	1	112	101	11	—	—	—	203	191	12
Dec.	32	32	—	37	37	—	41	37	4	—	—	—	110	106	4
Total	59	59	—	177	176	1	317	301	16	30	28	2	583	564	1

Klodnitzki's figures are not quite complete but they give an interesting insight into the monthly distribution of plague.

In 1910 plague was observed in at least 13 localities, some of which had been formerly attacked. No detailed accounts as to type have been published, but pneumonic cases were certainly observed<sup>(296)</sup>, especially at Tolubai-Dshapalakti, Beketai, Ak-Balik<sup>(297)</sup>.

(295) Russki Vr., 1913, No. 30-31.

(296) L.G.B.R. 1910-11, p. 167.

(297) Shurupoff, R. Vrach, 1911, pp. 1097-1106.



In 1911 many localities were affected. Deminski<sup>(298)</sup> mentions 49. Some outbreaks were traced back to camels. Up to 1911 all reports about camels as carriers of plague were unconfirmed and the experimental evidence was contradictory, if not negative<sup>(299)</sup>.

For 1911 four instances are quoted<sup>(300)</sup> where B. P. was found in camels:

a. Saganai:	Camel belonging to sick family	} Both animals showed mix. infect. at p.m.
b. Aktshagil:	" " " " " "	
c. Akshota:	" killed on Nov. 20; hum. c. fr. Nov. 24. Man, who cut the meat, rem. healthy .....	} Both animals pos. for B.P.
d. Sari-Tube:	" stat. to be respons. for one pl. case (anim. was killed, when sick). Human c. not con- firm. bacteriol.....	

The above evidence is not particularly strong to suggest this new theory. Later on a more likely case will be reported. The occasional part played by the camel was again referred to in 1923<sup>(301)</sup>.

The year 1912 marks an epoch in the history of plague in these regions because it was then proved finally that wild rodents of the steppes are responsible for frequent human outbreaks. It is much regretted that these results were obtained at a dear price, for Deminski, one of the ablest workers in this area, became infected, while performing a *post mortem* on a suslik<sup>(302)</sup>. He was attacked with pneumonic plague, both he and his pupil Krassilnikova, who nursed him, succumbing to the disease.

The usual outbreaks were bubonic:

- a. Rachinka, Aug. 20 cases with 16 d., preceded by epizootic.
- b. Dshanibek, Aug. 6 " " ? "
- c. Savjetnoe, July 36 " " 25 " (303)

In 1913 8 outbreaks claiming 45 cases with 40 deaths were counted. Among these were 9 fatal pneumonic cases, occurring in June at Solokhin (Tsaref district) as well as well as 11 pneumonic and 2 bubonic cases in June at Shitkura. On this

(298) Vj. Guig., 1912, p. 1329. L.G.B.R. 1912-13, p. 71.

(299) Klodnitzki, Astr. Conf. Rep., pp. 110-111; Bull. Off. Intern. d'Hyg. Publ., 1912, 3, pp. 431-480; Shurupoff, R. Vr., 1911, No. 52.

(300) Vj. Guig., 1912, p. 349; Deminski, *ibid.*, p. 1329; Klodnitzki, R. Vr., 1913, No. 30-31.

(301) Zabolotny, Ann. Inst. Pasteur, 1923, June.

(302) Klodnitzki, l. c. (1913).

(303) *Ibid.*

occasion Dr. Theodorof died suddenly. He had been employed in the anti-plague service and it was suspected that he died from plague<sup>(304)</sup>.

In 1914 56 cases with 46 deaths were recorded up to August. Dr. Jesimoff received infection from a patient and died of pneumonic plague<sup>(305)</sup>.

From 1914 to 1921 no information was forthcoming. From private sources it was ascertained that there was no marked recrudescence.

From July 1921-Febr. 1922 23 plague cases were observed in the Kirghiz steppes in 3 families at *Karamija* (Talovka district)<sup>(306)</sup>:

Bubonic—18; pneumon.—3; pn.-bubonic—1; skin pl.—1; total—23.

From Dec. 1922 -Febr. 1923 116 pneumonic cases were counted in two of the six governments of the Kirghiz Republic<sup>(307)</sup>.

The latest information to hand (April 1924) shows a total of 473 cases with 435 deaths for the first three months of 1924. The epizootic among wild mice is stated to continue.

*General remarks about plague in the Astrakhan Territory:*

The problem of the epizootics has been fully dealt with in our article on Wild Rodents. Some aspects of the human outbreaks, however, seem called for.

- i. There are few plague areas which can compare with Astrakhan in the rapidity with which plague assumes the pneumonic form. Our records amply bear this out. It can be seen that in all instances, where detailed information was available, the initial cases in the pneumonic outbreaks were either of the bubonic or septicemic forms. On the other hand, Zabolotny in his recent publications<sup>(308)</sup> presumes the existence of initial pneumonic cases and explains them by direct infection through the upper respiratory tract. It is not clear, whether Zabolotny had within recent years actually seen primary pneumonic cases due to such direct infection, or whether his statement is based on merely theoretical grounds.

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(304) Zabolotny, l. c.; L.G.B.R. 1913, p. 59; Clemow, Lancet, 1913, Nov., pp. 1575-77.

(305) Clemow, Lancet, 1914, Oct., pp. 911-913.

(306) Dobreitser, Hyg. and Epidemiol., Moscow, 1922, No. 1, p. 92; Nikanoroff, Rev. Microbiol. et Epidemiol., Saratov, 1922, p. 76.

(307) U.S.P.H.R., June 29, 1923.

(308) Ann. Inst. Pasteur, 1923, June, p. 624; Arch. f. Schiffs-und Tropenh., 1922, p. 382.



- ii. In view of the possible connection between the situation of buboes and the incidence of pneumonic type as contemplated by some authors, the following statistics from Astrakhan, though limited, may be helpful:

5 mainly bubon. outbreaks showed:		<i>Initial cases in 6 outbreaks:</i>	
Cervical buboes .....	11	Cervical buboes .....	3
Axillary .....	13	Axillary .....	2
Femor. and inguin. ....	39	Tonsillar pl. ....	?1
No buboes .....	2		
Pneumon. pl. ....	5		
Undeterm. ....	6		
<hr/> Total 76			
			<hr/> Total 6

- iii. The Kirghiz steppes have a continental climate with small atmospheric precipitation. The humidity is said to diminish progressively; its evaporation is considerable, especially under the influence of frequent dry south-eastern winds. The mean temperature is 7-8.5° C., the maximum being +45° and minimum -36° C. The winter is thus rather cold; the air is pure during the cold season when one of the frequent snow-or rather sand-storms is not raging. The heat in summer is suffocating. Respiratory diseases and lung tuberculosis are rare among the Kirghese<sup>(309)</sup>.

- iv. The houses of the Kirghese (principal sufferers of pneumonic epidemics) are generally of a poor quality; only the wealthier ones can afford to have floors and ceilings made of plain boards; the majority have to be satisfied with earthen floors, while ceiling and roofs are made from reeds. The walls are usually built of unburnt bricks, but the poor people use only reeds. The extreme poor dig an underground hole and merely cover it with a reed roof, which contains the window. (This is also seen in Siberia and N. Manchuria).

The huts are dark and insanitary and are not protected against the cold. As long as they heat their stoves (which are provided with a chimney, but no damper) it is warm, but as the heat is used

(309) Konstanzoff. Vj. Guig., 1902, pp. 1491-1515; Wolferz, Astr. Conf. Rep., pp. 149-180.

at the same time to keep the kettle boiling, the air is invaded by steam. Cold reigns when the fire is not regularly fed with cattle dung, especially as leakages occur everywhere. Naturally the steam condenses.

The huts are overcrowded; it would seem that the figures of Wolferz (108-216 cub. ft. of space and 11-16.5 sq. ft. of floor) represent a good average for each person. Bjelilovski said in 1910 that 10-15 persons lived on 98-147 sq. ft.

It must not be forgotten that all the above applies only to the winter huts and conditions prevailing during the cold period. In summer the Kirghese live either in their *kibitkas* (waggon) or in summer huts. Though pneumonic epidemics occurred generally in winter, there were exceptions. Hence it would not be wise to look upon the above mentioned factors as constituting the sole cause of pneumonic epidemics.

- v. The habits of the Kirghese are favourable for the spread of infection throughout the year. They hardly wash themselves and wear their garments till they wear out. Neither clothes nor caps are removed when going to rest. They use no furniture beside bedsteads which are placed against the wall in day time. Thus they squat on the floor covered with felt. They eat with their fingers; when the meal is over they wipe the mouth and fingers with a common towel; this is done also when guests are present and it is an honour to the host to possess a dirty towel, because this shows that he entertains lavishly.

The patients are visited by many visitors who often wipe the sputum of the patients with their fingers and then garments. The dead are washed by their friends, and many take part in the burials. The belongings of the deceased are eagerly divided. Memorial feasts are held for them, mainly on the 7th and 40th day after their demise.

Parasites abound in the huts. Fleas and lice are present everywhere, while bugs appear less prevalent.

These conditions are rather favourable for the spread of infectious diseases and the only antagonistic factor, the sparseness of the population (3.8 inhabitants per square verst) is counteracted by the readiness and speed with which the Kirghese travel on horseback to visit their friends.



## 2. URAL TERRITORY.

The first plague outbreak of modern times is described by Klimenko<sup>(310)</sup> and Filaretov<sup>(311)</sup> and started among the Kirghese inhabiting 4 hut between the localities Jamanchalinski and Saraitshikovski on Nov. 1, 1904. The epidemic lasted till Jan. 7 thus:

Locality	Huts inf.	Cases	Mode infection
4 huts .....	4	46	Original focus.
Vicinity .....	?	9	?
Saraitshikovski .....	94	216	Through a Cossack visiting his shepherd, who had been inf. in original focus.
Sorotshinski .....	15	52	Through persons assist. at funerals in Saraitsh.
3 auls near Jamanchalinski .....	?	17	Persons taking part in funerals of first victims.
Kirghese settlement Jamanchal.....	?	32	Persons taking part in funerals of first victims.
Jamanchalinski.....	12	26	Feldcher, infected at p.m.
Vicinity of Gurief.....	?	17	Kirghese, flying fr. aff. locality.
Grebentshikovski.....	1	1	Soldier, returning fr. Saraitshikovski.
Total Over 130 416 (all fatal).			

The epidemic was stated to be purely pneumonic and this holds true for the later cases. The early cases were apparently not seen by any medical man.

About 20 families each of 8 or more persons died out. The toll among the sanitary personnel was heavy. The medical assistant had apparently received no wound at the p.m., but was infected in the plague area. His death was followed by his wife, 3 children and three attendants. At Saraitshikovski all the burial personnel (17 in number) died. The panic stricken population then adopted drastic measures by sending the patients out into the cold out-houses.

Serum was given intravenously in considerable doses, but without result<sup>(312)</sup>.

The second outbreak on record at *Kis-Molla* (1907) where Sept. 14-19 3 men and 8 women died after killing a sick camel, was probably due to anthrax. In 1909 a summer outbreak of pneumonic plague was noted, in which four *kibitki* (canvas covered waggons) at Iltok were attacked:

(310) Astr. C. Rep., pp. 64-67.

(311) Vj. Guig., 1905, p. 683

(312) Klodnitzki, Astr. C. Rep., p. 460.

	<i>Persons :</i>	<i>First case :</i>	<i>Died :</i>
K. 1 .....	10	June 18	10
K. 2 .....	4	July 2	4
K. 3 .....	7	„ 11	7
K. 4 .....	7	„ 3	1
	—		—
Total .....	28		22
	—		—

The first cases were not seen by the medical staff. The fourth outbreak was recorded in November and December 1909 as follows :

<i>Locality :</i>	<i>Huts aff. Inhabit. Died. Healthy.</i>			
<i>Aul</i> 15, Tshedirinski distr....	1	4	1	3
„ 4, Dshambeitinski distr.	3	19	18	1
„ 5, Dshambeitinski distr.	28	170	121	49
„ 8, Ulentinski distr. ....	15	73	56	17
	—	—	—	—
Total .....	47	266	196	70
	—	—	—	—

This was pneumonic with exception of a few bubonic cases.

We have no detailed notes for 1910 (189 cases with 179 d.) when cases occurred throughout summer and winter<sup>(313)</sup> or for 1911, when isolated cases appeared<sup>(314)</sup>.

Plague continued to appear in sporadic form in 1912<sup>(315)</sup>. An interesting instance concerned a camel, where cases started in a family, *after* the sick camel had been killed, divided and eaten. Part of the meat was transported to a place several miles away and here one person also fell sick with pest. The camel could not be examined, but the nature of the human cases was fully established<sup>(316)</sup>.

In 1913 a severe outbreak occurred in October and November at Issim-Tjube and adjacent localities. Zabolotny<sup>(317)</sup> records 404 pneumonic and 2 bubonic cases. Koltzov found in Djambeita a few wild rodents plague affected at the same time as human cases.

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(313) Gray, l. c., p. 12.

(314) L. G. B. R., 1912-13, p. 72.

(315) Ibid. ; Clemow, Lancet, 1913, June, p. 1697.

(316) Deminski, Vj. Guig., 1912, p. 1329.

(317) Ann. Inst. Pasteur, l. c.



In March 1914, Inder (the centre of the 1913 epidemic) was again visited. 17 fell sick and 16 died; almost all suffered from pneumonic plague<sup>(318)</sup>.

Nikanoroff<sup>(319)</sup> recorded an outbreak in 1915 which was preceded by an epizootic among both wild and *domestic* mice; *Mus norvegicus* was also found infected. The spread of the infection to domestic rodents had hitherto been unobserved in those parts.

### 3. DISTRICT OF SAMARA.

In September 1899 an outbreak "of malarial fever complicated by secondary infection affecting the intestinal canal and associated with pneumonia" was reported<sup>(320)</sup>. Such diagnosis was quite common at the time to express plague in vague terms<sup>(321)</sup>.

### 4. DON PROVINCE.

50 cases and 16 deaths were reported (315) in Popov and two other villages in November and December, 1912. The outbreaks were suspected to have been caused by susliks, which had lately much increased in numbers in those parts, owing to the extermination of the foxes.

Retivov states in a study of the 1913 outbreaks<sup>(322)</sup> that bubonic cases (42 with 24 d.) had been noted in the summer of that year. The susliks were undoubtedly responsible and several cases had been observed among them, some children being directly infected. He also refers to a *migration* of susliks from the Astrakhan Province in the Don Territory. Retivov's figures for autumn 1913 are:—

<i>Locality :</i>	<i>Started :</i>	<i>Ended :</i>	<i>Cases :</i>
Novopetrovsk .....	Sept. 17	Oct. 17	35
Chutor Kalatsh .....	,, 27	,, 15	5
Chutor Breslavsk .....	Oct. 9	,, 21	6
Chutor Gromoslavsk ...	,, 7	,, 23	13

Total ..... 59

(all pneumonic, fatal).

(318) Clemow, Lancet 1914, Oct. 10, pp. 911-913; L. G. B. R. 1914-17, p. 147.

(319) Rev. Microb. and Epidem., Saratov, 1922, pp. 71-72.

(320) L. G. B. R. 1898-01, p. 123.

(321) Original Home of Pl., Hist. Evid.

(322) Vj. Guig., 1914, p. 674.

## Age and sex incidence:

1½—5 yrs.	5	41—45 yrs.	3 (1 m., 2 f.)
6—10	3	46—50	8 (2     6 )
11—15	3	51—55	1 (1     0 )
15—20	5 (3 m., 2 f.)	56—60	6 (2     4 )
21—25	3 (2     1 )	61—65	1 (1     0 )
26—30	8 (5     3 )	66—70	1 (0     1 )
31—35	2 (1     1 )	71—75	1 (1     0 )
36—40	6 (3     3 )	Undeterm.	3

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Total 59 (22 males, 23 fem., 11 ch., 3 undet.)

One of the first victims was a medical assistant, who had attended a patient on Sept. 25. He returned home on the 27th and was admitted to the general ward of hospital. He died, followed in quick succession by another feldcher, his wife, the hospital guard and a nurse. The panic-stricken patients (10 in number) ran away from the hospital; two of them were plague infected and spread the disease. In the later course of the epidemic two other feldchers succumbed to plague.

## X. PNEUMONIC PLAGUE IN CENTRAL ASIA.

### 1. TRANSCASPIAN PROVINCE.

Two early outbreaks—one in Askabad in 1892, called 'Black Death' in the report of the Governor and killing in six days 1300 out of a population of 30,000, the other in and near Merv and causing in 1896 (Sept.-Oct.) 10,000 deaths<sup>(323)</sup>—are somewhat suggestive of pneumonic plague. Both accounts are indefinite.

An undoubted outbreak of pneumonic plague is reported in Dec. 1912 from the *aul* (village) Tshuiruk, 8 miles from Merv and killed 52 persons. The disease was said to have been introduced by smugglers from Khorassan. Elaborate measures, including a cordon, were taken and the outbreak was confined, though there were rumours of 96 cases in Merv<sup>(324)</sup>.

### 2. TURKESTAN.

Only one outbreak with details is recorded<sup>(325)</sup>, in which pneumonic cases predominated. This occurred in Sept.-Oct. 1898 at Anzob, a village lying 7000 feet high in the Hissar mountains (167 miles south from Samarkand).

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(323) L. G. B. R., 1879-98, p. 246-247.

(324) L. G. B. R., 1912-13, pp. 72-73; Zabolotny, Arch. f. Schiffs- & Tropenh., 1922, p. 380.

(325) Levin, Vrach, 1898, p. 157.



The disease started at the end of August in the village Marsich, 11 miles away. One of the patients was visited by a female relative from Anzob, who returned home after the death of the patient. She fell sick and the disease spread among her relatives and other contacts, sparing neither adults nor children.

During the first month 224 fell sick with 219 deaths in a village of 387 inhabitants. Only three out of the 60 families were unharmed. In October it abated and claimed 29 cases with 18 deaths. Levin states that at the beginning of the epidemic the pneumonic form was prevalent, then mixed cases and finally purely bubonic ones (mainly inguinal buboes).

### 3. SEMIRETCHINSK GOVERNMENT.

In our article on Wild Rodents we mentioned the outbreak occurring in 1907 (Aug.-Sept.) on the Aksai plateau (Atbashinsk district), caused by a black marmot. The man who caught this animal, soon succumbed; 12 members of his family and 14 others in the same settlement shared his fate. A woman, survivor, brought the infection to another settlement, where 19 more perished. This outbreak, which was bacteriologically confirmed, was pneumonic<sup>(326)</sup>.

The same type was again manifest in July and August 1910 in the district of Prjevalsk<sup>(327)</sup>.

In 1911 and 1912 only few cases seem to have been noted in autumn<sup>(328)</sup>, the nature of which is uncertain.

In September 1913 there was another pneumonic outbreak among the Kirghese in Tourgouen with 31 cases (all fatal except one)<sup>(329)</sup>.

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## XI. PLAGUE IN TRANSBAIKALIA, NORTH MANCHURIA AND MONGOLIA.

As in the case of Astrakhan and adjoining territories, a discussion of bubonic as well as pneumonic outbreaks in these regions is called for.

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(326) Russian Publ. H. Rep., 1907, p. 162.

(327) L. G. B. R., 1910-11, p. 167; Young, Shansi Pl. Rep., 1918, p. 2.

(328) L. G. B. R., 1912-13, p. 72.

(329) Ibid., 1913, p. 60.

## 1. TRANSBAIKALIA AND NORTH MANCHURIA.

The first *record* of an illness suggestive of plague dates back to 1863. According to Dudshenko-Kolbasenko<sup>(330)</sup> an acute fatal disease raged in that year in the village Zagan-Oluevski (Transbaikalia), beginning during the hay harvest in August. The first victim is said to have hunted, skinned and eaten tarabagans. The outbreak must have been rather virulent, for some families were entirely wiped out. It terminated in October. Dudshenko states that the fourth of October is still celebrated in this village as a thanksgiving day.

The next invasion occurred in 1875<sup>(331)</sup>: According to a Buriat bone-setter, his father, living 13 miles from the Borzia river, was called to attend some patients, who had fallen ill after eating 'diseased' marmots. Four of those patients died, as well as the Buriat attendant, his wife and four other persons. The man himself fell sick, but recovered and could not remember his symptoms.

The records of the next manifestation in 1880<sup>(332)</sup> are clearer. In the village Klitshki (Nertshinsk District) a peasant fell sick after having eaten meat from a dead tarabagan. He showed cervical buboes and died on the 5th day; his brother and sister were soon afterwards attacked with inguinal buboes, but recovered. When the brother was convalescent he went to the bath-house in company with another man, who later developed fatal bubonic plague.

1885: A doubtful outbreak at Kulusutai is recorded by Kashkadamov<sup>(333)</sup> and Skchivan<sup>(331)</sup>, where a whole family of 7 persons died.

1888: A. Bjeliavski<sup>(334)</sup>, Rjeshetnikoff<sup>(335)</sup>, Favre<sup>(336)</sup> state that in Barachol, 27 miles from Kulusutai, 5 Buriats died in one *jurte* (tent); it is asserted that they were infected by tarabagans. Ashman and his feldcher Judin were sent from Aksha to investigate these mysterious deaths. They performed p.m. on two dead bodies and diagnosed 'typhus'. Two days after (Oct. 18) the feldcher fell sick, developed a left axillary bubo and died on the 20th. The doctor, attacked one day later with bilateral axillary buboes, was brought home, where he succumbed on Oct. 24th.

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(330) Vj. Obst. Guig., 1909, pp. 1045-1089.

(331) Arch. Russes de Pathol., 1901, pp. 603-612.

(332) Besser, Vojenno-Med. Jl., 1906, Febr., p. 284.

(333) Vj. Guig., 1906, July-August.

(334) Ibid., 1895.

(335) Ibid.

(336) Z. f. Hyg. & Inf.-Kr., 1899, May.



B. At Ulus Kungur in the same district 9 Buriats in 2 *jurtes* died.

1889: At Sektui there was 'pestilence' in June among the tarabagans. In July a girl (aet. 15) of a family of hunters fell sick and died on Aug. 2; the next death occurred on the 19th in a woman relative. Summarising, the cases are:—

In the first family one girl, 2 men, 1 woman died, 6 sons remained.

In the second family 2 men, 1 woman, 2 children died, 1 child remained.

All patients had buboes, mainly axillary and inguinal.

Furthermore a Tunguse boy, who used to play with the children, died. Cases were also present among the Buriats near the village<sup>(332)</sup>. This was probably not the first visit of plague at Sektui. Wassilevski<sup>(337)</sup> states that the old Buriat name for the village meant 'infection'.

1890: Kashkadamov<sup>(333)</sup> notes the total extinction of a Buriat family at Haranor.

1891: A. At the end of September the dog of a Cossack brought a dead tarabagan into the court yard of his master; one of the sons skinned the rodent and gathered the fat. He and his brother fell sick with bubonic plague. Their family was quarantined by the neighbours, and no more cases developed.

B. A Cossack at Aksha visited a shepherd in Mongolia on Sept. 3. On the way he shot and skinned a tarabagan and ate some of its meat. The wife who prepared the meat and partook of some, died on the 10th. The Cossack died after returning home Sept. 9. Thirteen fell sick and six died in this family; a man who took part in the burial and his wife were fatally infected, while Dr. Utkin and feldcher Savatjev, who attended the patients, recovered from slight attacks. Rjeshetnikoff<sup>(335)</sup>, who assisted in the treatment of the patients, diagnosed *pneumonic* plague, emphasizing that in none of them could he see buboes. His statement is questioned partly because Utkin found inguinal buboes in at least one patient. There is no doubt, however, that some of the patients had bloody sputum and therefore secondary if not primary lung plague.

1894: A new outbreak occurred in September at Sektui. It appears that the dog of a Cossack, journeying from Zagan-Olui into Sektui, brought 6 tarabagans to his master in a

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(337) Vj. Guig., 1915, pp. 178-201.

short time; the animals were certainly sick, otherwise they would not be so easily caught. The Cossack himself and six members of his family died within a month, apparently from bubonic plague. Strict measures undertaken by the neighbours prevented further spread. This same outbreak is discussed by Rjeshetnikoff in a second paper<sup>(338)</sup> as occurring at Aksha in 1895. The dates of different authors regarding the early outbreaks differ sometimes; in this instance, however, it seems certain that no outbreak occurred from 1894 to 1903. Perhaps small manifestations were overlooked<sup>(337,339)</sup>.

1903: 4 or 5 Buriats died in the vicinity of Zagan-Oluevski with symptoms suggesting plague after eating marmot flesh<sup>(333,330)</sup>.

No human outbreak is on record in 1904, but Kashkadamov affirms that a big epizootic raged in that summer near the lakes Chindan and Zagan-Nor.

1905: This year is memorable in the plague history of Transbaikalia and North Manchuria, because the nature of the human cases was first bacteriologically established.

The outbreaks near the Siberian railway (completed in 1900) are described by several observers, the evidence being augmented by expeditions to the steppes. The result of the joint investigations<sup>(333,340)</sup> shows that epizootics raged among the tarabagans at several points, and were responsible for quite a number of human outbreaks. It is surprising that the infection did not spread among man, as all the preliminary conditions for a big epidemic seem present.

Marmot disease was reported by the natives in the following localities:

- A. In a large area near the road leading from Hailar to Old-Zuruchaitui on the Argun river, extending eastwards near to station Jakoshe. An epizootic was observed in July, especially at Chuduk and Chadatui.
- B. Near Abagaitui (17 miles north of Manchouli).
- C. Near Klutshévski, south of Haranor station.
- D. Between Chindant and Sharasun.
- E. On the Salt lake 33 miles south-west of Manchouli station.

The following human outbreaks can be traced to area A. or B.

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(338) Vratsh. Gaz., 1908, pp. 207-208.

(339) Tshausoff, R. Vratsh, 1911, No. 24-25.

(340) Chmara-Barshevski, Pl. Rep. of the Chin. East. Railway, Suppl.; Klodnitzki, R. Vratsh 1905, pp. 1595-1599; Korentshevski, *ibid.*, pp. 1497-1499.



## i. Dalainor village with one case at Manchouli.

It appears that the first victim, a Cossack (aet. 24) had visited the hay harvests east of the Argun river. He fell sick on Aug. 15 and died after 4 days with axillary bubo. The disease spread among his family and other contacts as follows :

Axillary bubo—2 ; inguinal—6 ; ing. and axill.—1 ; undeterm.—4 ; total—14.

With one exception all cases occurred between Aug. 15 and Sept. 13, when the whole population of 150 were bathed, clad in new garments and quarantined in railway waggons. Only one case (with lung complications) developed on Sept. 15.

No epizootic among domestic rodents was seen. The people even stated there were no rats or mice in their houses.

ii. The Mongols living in this area realised the danger of tarabagan pest and shifted their camps. One group, decamping to the Mutnoi-River (tributary of the Argun) escaped too late, for some fatal plague cases occurred among them.

iii. An expedition made by Korentshevski and Klopfer in the district of Solons (former Mongolian territory now included in North Manchuria) reported an outbreak affecting four *jurtes* in July and August 1905. The first victim was a young shepherd working in the worst-infected zone (Chuduk and Chadatui). On the way home in July he caught, prepared and ate an apparently sick tarabagan. Sickness developed, characterised by cervical and axillary buboes and lasted for 20 days. Towards the end bloody sputum appeared and he died. All the other cases can be traced back to this. Tabulation as follows:—

Jurte	Persons	Died	Healthy	Remarks
1.	6	4	2	
2.	7	1	6	
3.	5	3	2	First pat. had buried corpse at j. 1.
4.	5	3	2	„ „ was lama, attended j. 1.
Total	23	11	12	

All the victims had buboes (axill. and cervical) ; many showed lung complications.

No human cases were reported from the area *C*. In area *D*, however, both epizootic and human cases were seen:

- i. Kashkadamov records the death of 2 Buriats in Zaganorski Tsan-Kondoi in August.
- ii. 8 miles from Chindant a shepherd died in September after 3-4 days illness, followed in quick succession by his son and the *shaman* (sorcerer). Another shepherd, burying the dead bodies, also fell sick with *bloody sputum* and died in 3 days.

*Area E*. Besides several suspicious cases, one undoubted plague case was observed in an inhabitant of Manchouli who fetched salt from the lake. He fell sick on Sept. 11 with axillary bubo and died three days after.

*1906*: The epizootic noted in 1905 near Abagaitui raged again in 1906. A Cossack in this village, suffering from tuberculosis, craved for tarabagan meat which he considered excellent for nourishment and as a remedy. His dogs brought him ten marmots. He fell sick on Sept. 23 and died after 4 days, followed by his wife, relatives and friends in two other houses. Up to Oct. 18, 6 (2 men, 3 women, 1 child) were attacked.

A military orderly who attended some patients, reported sick on Oct. 19th and died after 2 days in the Manchouli Hospital. Two more cases followed, one a youth at Abagaitui and another a soldier at Manchouli, both contacts to the orderly.

It seems that the outbreak was mainly *pneumonic*<sup>(341)</sup>.

*1907*: In 1907 Barikin succeeded in establishing *prima facie* evidence of plague in a tarabagan shot by him in a suspected locality. His findings are the more important because not far from this locality there had occurred a fully confirmed plague case in a girl (æet. 13). All details in this connection are contained in the paper on Wild Rodents.

Of equal importance is the history of a woman, æet. 47, falling sick with axillary bubo in Substation 83 on the Transbaikalian Railway on Sept. 4-7. This patient had not been in the fields, but was engaged in the tarabagan skin trade. Our latest investigations prove the possibility of infection through skins. We have personally seen in 1923 ectoparasites, including fleas, on marmot corpses and also on moist and dried skins.

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(341) Barikin, Russki Vr., 1909, pp. 538-540.



A third fatal bubonic case, occurring in Sept. 1907, 3 miles from Haranor was also due to tarabagan hunting.

1908: i. Some suspicious cases were reported in the middle of August at Sun in Transbaikalia<sup>(342)</sup>.

ii. Dudshenko<sup>(330)</sup> relates that in Kulusutai (south of Borzia) a Cossack of Buriat extraction died of septicemic plague on Sept. 25 and that through his relatives the infection was carried to Mongolia.

1910: This historic year is connected with the great Manchurian epidemic which swept over Manchuria and North China as far as Peking and claimed 60,000 lives. It began quietly in the sparsely populated tarabagan regions of Transbaikalia killing a few persons, and attracted little attention until it reached Northern Manchuria where the Chinese Eastern Railway conveyed the infection broadcast. The later course of the disease has been voluminously described in several publications of various languages. Our duty here is to concentrate more upon the early phases.

When studying the history of plague two main methods of spread are discernable, namely (i) quick method through some *extraordinary* event such as, war, famine, earth-quake or other catastrophe; (ii) slower or *peaceful* method, depending upon usual conditions of traffic and commerce. In the first Manchurian epidemic we notice the operation of this peaceful means of spread. The increasing demand for tarabagan skins in the world markets attracted a large host of hunters to formerly unfrequented districts. The prize of a marmot skin, quoted at rouble 0.30 in 1907, had risen to rouble 1.20 in 1910; 700,000 skins were exported from Manchouli in 1908, 2½ millions in 1910. This large supply was produced not so much by an increase of hunting as by an increase in the number of hunters. Not less than 11,000 Chinese hunters were counted at Manchouli in summer 1910, and in October of the same year, 4600 of them were still present.

The native Buriats and Mongols possessed ancient traditions and long experience, which taught them to kill the animals by shooting, so that they were able to judge whether their prey was healthy or not. A sick tarabagan would be sluggish or lame and would be left alone by them. Parties notified one another of the infected spots so that they might avoid them. When, notwithstanding all precautions taken, the disease broke out, they recognised it immediately and took drastic measures, such as leaving the sick alone, removal of quarters, etc.

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(342) Pisemski, in Chmara-Barshevski, p. 64.

This example was followed, though quite unmethodically, by the Russiass, who had settled down to the business.

In the case of the Chinese, it was different. The newcomers were usually raw and ignorant coolies from the villages of Shantung, who had never seen a tarabagan in their lives. They knew no history and had never heard of plague among the animals. Hence they caught tarabagans indiscriminately with snares and even congratulated themselves when they saw a sluggish one. One sick animal might provide the spark for an epidemic. Inexperience and overcrowding in underground inns would supply the necessary fuel for its spread. This state of affairs, slow in its evolution, was not grasped by even the educated, and thus disastrous consequences resulted in 1910.

The following data of epizootics and early human outbreaks have been collected:

- A. On July 1 a sanitary commission in Aksha discussed a telegram of the Ataman of the Duroevski Stanitza, reporting 'tarabagan disease' among natives near the river Gan (right tributary of the Argun). This news was twice confirmed, but no preventive measures were undertaken.
- B. A party of hunters in the service of a certain Popov (baptized Chinese) put up in August two tents on the eastern shore of the Argun, 30 miles from Kailastui. Towards the end of August some disease broke out among them and when the master visited the camp in middle of September, he found that:

in one tent had died .....	4 out of 5 inmates.
in the second .....	3 ,, ,, 6 ,,

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Total ..... 7 out of 11 inmates

The remaining four had fled to Manchouli and could not be found<sup>(343)</sup>.

- C. The interpreter of the Chinese frontier post Kailastui reported that at the end of August four Chinese had died in quick succession. All showed *bloody sputum*. (These four persons might be the same as these reported to have escaped to Manchouli.—Ed.).

Chmara-Barshevski (Chief Sanitary Surgeon of the Ch. Eastern Railway) who investigated the matter, thinks it probable that the disease at Manchouli commenced, not on Oct.

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(343) Malov, in Chmara-Barshevski, l. c.



12, as officially stated, but early in September. There was also private information that at the beginning of September some Chinese had died suddenly with cough and bloody sputum. The contacts ran away, and some probably died. Our own investigations showed many cases in the Chinese inns in late September.

D. The demand for tarabagan skins also affected Hailar city on the Chinese Eastern Railway. Up to 1908 this place played no important part; but in 1909 400,000 skins were collected there and in 1910 nearly a million was supplied by 5000 hunters. These men hunted near the Ugunor lakes on the rivers Emengol and Mergen, a district avoided by the Mongols in 1916, because in April several of them, hunting near the Mergen lake and river had been struck fatally. Over 20 Mongols then died. Vodakoff, Chmara-Barshevski's informant, was warned by the Mongols to avoid some infected tents, where Chinese hunters had died. These reports were confirmed from other quarters, as well as the news of an epizootic near the river Emengol. It is thus quite possible, according to Chmara-Barshevski, that Hailar was infected directly from the adjacent steppes<sup>(344)</sup>.

E. The evidence brought forward at the Mukden Conference<sup>(345)</sup> may now be taken. Ch'uan, sent by the Chinese Government to investigate, reported that several Chinese carpenters had died with pneumonic symptoms in Dauria (Transbaikalia) on September 16th and that plague was imported from there into Manchouli. As this neighbourhood abounded in tarabagans, it was quite possible for the prevailing epizootic to affect a number of foci where these thousands of hunters earned their living. Gray also recorded suspicious cases in Aksha, probably of marmot origin.

Many cases, occurring in late September were hidden from the authorities and thus not reported officially. By the middle of October pneumonic plague had been well entrenched at Manchouli. Here the first case was diagnosed on Oct. 12 in a house inhabited by tarabagan hunters, where 14 died out of 40. Only one patient, a hunter, aet. 35, was found, the contacts having all escaped. P.M. performed on the 13th established the diagnosis of pneumonic plague.

Up to Nov. 12 there were found 158 sick and 72 corpses. On this day the railway authorities decided to evacuate the

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(344) L. c., p. 92.

(345) Ch'uan Shao Ching, Conf. Rep., p. 27; Gray, *ibid.*, p. 192.

majority of the poor Chinese population, among whom the plague was rampant. Over 3000 persons were housed in goods waggons, each holding 10-15, early on the 13th and kept there until Dec. 9. This evacuation, carried out with all sanitary precautions, produced excellent results. From Nov. 13 to 28, when the last case occurred, only 53 cases were reported.

The patients altogether at Manchouli were:

Chinese .....	284, died 284	Total of patients .....	294
Russian .....	10, ,, 9	,, corpses .....	342?
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Total .....	294, died 293	Grand total .....	636?

One case, alleged to have recovered after 12-13 days, was a child (aet. 3) whose parents had died of plague. He was supposed to show physical signs in the lungs<sup>(346)</sup>, but as no plague bacilli were seen at any time, the case seems unproved.

No details for the various affected localities are here given, because reference can be made to the Mukden Report of 1911.

The epidemic spread from Manchouli westward back into Siberia and eastward into China. From Tsitsikar it branched northwards as far as Mergen, halfway between Tsitsikar and Taheiho (Aigun). Harbin owing to its strategic situation soon became the central focus of infection. From here it continued eastwards as far as Hantaohotzu but did not reach Suifenhö or Vladivostok. Southwards it carried death broadcast attacking Shwangchengpu, Changchun, Kirin, Mukden, Shanhai-kwan, Dairen, Tientsin, Peking, Tsinan and Chefoo. Altogether, counting from Manchouli to Tsinan (Shantung) the pest travelled the long distance of 1700 miles. The time taken from the beginning (Sept.) till the end of the epidemic (April) was 7 months.

The approximate number of deaths was as follows<sup>(347)</sup>:

Heilungkiang Prov. ....	15,295
Kirin Prov. ....	27,476
Fengtien (Mukden) ....	5,259
Mukden-Port Arthur ....	76
Mukden-Tientsin-Peking .....	1,693
Peking-Hankow, Chihli Portion .....	173
Peking-Pukow, Chihli and Shantung .....	928
Shantung Peninsula ..	1,562
<hr/>	
Total (approximate) .....	52,462

(346) Chmara-Barshevski, p. 65.

(347) Chmara-Barshevski, l. c., Gray l. c., Peking 1911.



In Transbaikalia the number of cases, according to the Russian Railway Report, was:—

Dauria .....	12
Substation 37 .....	3
Petrovski Zavod .....	13
Total .....	28

These figures seem too small<sup>(348)</sup>.

The number of those who succumbed fighting the modern Black Death is not exactly known, but the following incomplete list is given:

Deaths among sanitary staff:

Locality	Grade	Deaths	Remarks
Manchouli.....	1 feldcher, 4 attendants (all Russian).....	5	Among 636 cases
Tsitsikar .....	Sanitary and other personnel .....	105	„ 2032 „
Harbin .....	Railway area: 3 doctors, 2 students, 5 feldchers, 1 Sister, 21 Russian, 7 Chinese attendants, 2 washermen, 1 Thibetan practitioner .....	42	
	Fuchiatien (Chinese): 1 doctor, 1 dresser, 4 native practit., 2 French priests, 53 police etc, 102 coolies, 4 cooks, 69 ambulance men, 63 soldiers .....	299	Out of a staff of 2943.
Shwangchengpu	100 coolies, 140 police ...	240	Out of 200 coolies and 270 police.
Changchun .....	18 native pract., 24 nurses, 21 burial coolies, 5 disinfecters, 22 emergency coolies, 30 grave diggers .....	120	Total number of personnel: 320 burial coolies, 156 disinfecters, 120 emerg. coolies, 91 grave diggers.
South Manchuria	2 doctors, 40 native practitioners, etc. ....	122	I. e. 2,66% of total cases.
Cihefoo .....	2 French Sisters.....	2	
Tientsin.....	2 doctors, 2 students, 1 midwife .....	5	Total cases 111.
Other places ...	Native practit....	2	(348)
Total		942	

(348) Mukden Rep., p. 32.

A moderate statement shows that one out of sixty deaths occurred among the sanitary, burial and other personnel.

A few remarks may be made about the types of plague met with during the epidemic. The 'septicemic' type, i.e. a form of plague with a course too rapid to develop marked lung symptoms, corresponding to the 'pulmonary' type mentioned in our Report 1918-21, perhaps played a not unimportant role; not sufficient attention has been paid to this, but some observers<sup>(349)</sup> mention it distinctly.

Though the contrary was stated occasionally, there is no doubt that *rare* cases of the *bubonic* type were met with; no definite statement was made at the Mukden Conference regarding their pathogenesis except in one case of cervical bubo reported by Fujinami where an infection from the fauces was presumed<sup>(350)</sup>. The buboes were not all cervical, as Murata quoted one in the femoral region<sup>(351)</sup>.

*Recoveries:* Besides the case reported from Manchouli mention must be made of the three instances discussed at the Mukden Conference by Abe. Plague-like bacilli were seen in their sputum (only microscopical examination). The attitude of the Conference towards these cases was very sceptical.

*Carriers:* i. The case of Mrs. Liu of Mukden attracted much attention at the Conference. This lady, sole survivor of a plague stricken family, infected to all appearances the inmates of all three houses visited by her. She herself remained healthy; neither her sputum nor her serum gave any indications of plague.

ii. Askanoff<sup>(352)</sup> found on four occasions plague-like bacilli in the sputum of apparently healthy contacts (2 Russian, 2 Chinese). Cultures from the sputum of all four and animal experiments with the sputum from one gave negative results. This evidence seems incomplete.

iii. A positive experimental result was obtained by Padlevski and Zlatogoroff<sup>(353)</sup> with materials swabbed from the mouth of a Chinese attendant. The inoculated gp. died of plague after 34 days. Unfortunately the attendant had left the service, as the epidemic was then at its end. Thus it is undecided whether

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(349) Christie, Mukden Rep., p. 166.

(350) Ibid., p. 151.

(351) Ibid., p. 150.

(352) Chmara-Barshevski's Rep., p. 317.

(353) Z. f. Bakt., Ref., Bd. 55, 1912, p. 354.



he was a healthy carrier or in the early stages of plague infection.

Perhaps one of the most extraordinary, and withal consoling features of the 1910-11 epidemic, was the apparent immunity of the native Chinese physician Ku (aet. 42) and his assistant Chia (aet. 28) who had charge of the plague house in Fuchiatien before the arrival of the modern medical staff. For weeks, day and night, these faithful men were at their post attending to the sick and dying, neither wearing a mask nor adopting any sanitary precaution, and yet they lived through this terrible epidemic. At least 1,200 patients passed through their hands, and while other attendants in the hospital died, these two men survived. They were in charge for six weeks, until the plague house was burnt down by order. It was asserted that Ku had had a slight attack of plague through accidental pricking of his finger by a needle which he had used for acupuncture upon a patient<sup>(35c)</sup>. His immunity might therefore have been acquired.

Not less marvellous perhaps was the example of Dr. Haffkine, who was in charge of the Russian plague hospital. Though he was in constant attendance upon the patients, he seldom wore a mask and yet came out of the epidemic unscathed.

1911: The great pneumonic epidemic ended in the early spring of 1911. Naturally it had drawn the attention of all concerned to the originally infected districts and to their rodent population. A number of expeditions—both Chinese and Russian—were undertaken in summer 1911. These obtained ample information on the subject<sup>(355)</sup> and found plague sick tarabagans in a few isolated instances.

These important findings are mentioned in the article on Wild Rodents.

Epizootics were located in the following areas:

- A. 2 miles from Sharasun (station of Transbaik. Ry., 28 miles fr. Manchouli).
- B. Near the lake Chabarda (16 miles south of Sharasun).
- C. Near post-station Arabulak (37 miles from Borzia on Transbaik. Ry., 76 miles from Manch.)

In September 1911 a small outbreak occurred in the village Nadarovsk,  $\frac{2}{3}$  mile from Sharasun station and was apparently

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(354) Mukden Rep., p. 232.

(355) N. Manch. Pl. Pr. Serv. Rep., 1911-13, p. 9.

connected with area A. The first victim, a Cossack, aet. 48, had been in the fields before he fell sick on Sept. 1. As no buboes were found, his case was probably septicemic. This patient infected:

His daughter .....	aet. 20	? septicemic
„ son .....	„ 14	cervical bubo
„ son-in-law .....	„ 26	cerv. and ax. buboes
and the latter's father		? septicemic
Total .....	5 cases	(16 other cont. rem. healthy).

Besides this outbreak two occurring in summer 1911 may be noted:

- i. Among the Mongols camping near the river Gan (see 1910), in mid June. 15 nomads and 2 lamas attending them died.
- ii. In Akra, near the Nertchinsk Factory (near the Argun river) at the end of June. Bubonic cases among nomads.

When the outbreak in Nadarovsk ended on Oct. 10, rumours began to circulate that there were suspicious cases in Dauria, where many Chinese were employed in the construction of military barracks. A medical commission visiting Dauria found out that there had been two suspicious cases among them; one of them had fever and bloody sputum. Both died on the way whilst being transported to Manchouli. Probably no plague was present, otherwise a big outbreak would have resulted among the others<sup>(356)</sup>.

The passenger traffic was then medically inspected at Manchouli. Only one plague case with tonsillitis was detected on Oct. 17th in a Cossack arriving from Dauria.

1912: Three fatal pneumonic cases were reported in late autumn among Cossacks who had eaten flesh of dead tarabagans<sup>(357)</sup>. These were confirmed by Haffkine<sup>(358)</sup>. No details are obtainable as to the exact location.

1914: A considerable outbreak occurred at Haranor<sup>(359)</sup>. Its source is apparently the Zasulan valley—the same locality where we and Sukneff found an epizootic in 1923. The first case occurred in a Cossack, aet. 53, who hunted at the end of July and died on Aug. 6. The spread of the infection is shown in two tables. All the cases except one were among relatives.

(356) Chmara-Barshevski, Suppl., p. 74.

(357) L. G. B. R. 1912-13, p. 73; Clemov, Lancet, 1913, June, p. 1698.

(358) N. M. Pl. Pr. S. Rep., 1911-13, p. 15.

(359) Wassilevski, Vj. Guig., Febr. 1915, pp. 178-201.



## (a) Age and sex incidence:

<i>Sex of att.</i>	<i>No.</i>	<i>Age:</i>
Men .....	3	53, 19, ?
Women .....	9	53, 37, 29, 19, 14; 4 undet.
Children .....	6	6, 5, 4, 3, 1½, 11 months
<hr/>		
Total .....	18	

## (b) Situation of bubo:

<i>Site:</i>	<i>No.</i>	<i>Remarks:</i>
Inguinal .....	10	1 with sec. pn. 1 recovd.
Axillary .....	6	1 with sec. pn. 1 recovd.
Cervical .....	2	
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Total .....	18	(3 recovd.)

Wassilevski believes that the infection, starting from wild rodents, was spread by human parasites which abound in the insanitary houses. In any case no infection could be proved in the local rodents though some attention was directed upon this problem. Rats were absent; domestic mice seemed quite frequent and apparently rat-hares also lived near the houses.

1916: Jetmar<sup>(360)</sup> reports two cases:

- i. A Cossack falling sick at end of August in Nadarovsk.
- ii. A Buriat shepherd dying about the same time in Turgo-Sungurskoi (Dauria district).

1919: A small outbreak occurring in September in Matsievskaja (12 miles from Manchouli on the Transbaik. Ry.) has been mentioned in the paper on Wild Rodents. Though the victims had handled tarabagans<sup>(361)</sup> it is more probable that they were infected from a dead hamster brought into the house by their cat<sup>(360)</sup>. One case showed right axillary and left inguinal buboes and another bilateral inguinal gland affection. Wassilevski<sup>(361)</sup> again points out the proximity of the holes of small rodents to the houses.

One suspicious case occurred in a Chinese tarabagan hunter 1½ miles from Matsievskaja; his dead body was removed by his friends so that no investigations could be made. This was suspected to be one of several concealed victims among the Chinese hunters<sup>(361)</sup>.

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(360) Jl. Transbaik. Med. Soc., Nov. 1922, pp. 1-13.

(361) Wassilevski, Jl. Harbin Med. Soc., 1921, No. 1-2, pp. 27-37.

1920: This is another fateful year as it marks the beginning of the Second Manchurian Epidemic. The brief notes<sup>(362)</sup> relating to outbreaks preceding it in the summer and autumn are now supplemented by fresh material at our disposal.

At the end of April it was reported from Haranor (Transbaikalia) that notwithstanding the cold tarabagans had vacated their holes. The inhabitants, on the lookout for plague, claimed to have seen corpses with suspicious signs. The Russian Plague Detachment<sup>(360)</sup> affirmed that they saw tarabagans leaving their holes. Snares were freely used for their capture. Neither in that nor other localities (Oloviannaja, Borzia, Manchouli) where epizootics were suspected, could sick animals be found. As we have pointed out<sup>(363)</sup> this early termination of the hibernation period does not necessarily mean any extraordinary event in the burrows. Furthermore, reports were current of a heavy mortality among marmots in early spring when food was scarce and enteritis frequent. Anyhow no human cases were noted during this spring hunting season.

Jetmar<sup>(360)</sup>, who studied most of the subsequent outbreaks, thinks that they correspond to at least four foci each independent of the others. These are:—

- A. Dauria (36 miles west of Manchouli on the railway line). There in the middle of September three soldiers were attacked almost simultaneously by fatal bubonic plague. Undoubtedly they were infected by tarabagans, much hunted with snares. The last case died on September 20. The patients showed axillary or inguinal buboes.

A girl, act. 13, fell sick with bubonic plague at the same time in substation 83 (8 miles distant from Dauria) and succumbed in the Oloviannaja Railway Hospital. She was probably infected from the same focus, though the mother denied any contact with tarabagans. She had axillary bubo.

- B. Abagaitui (on Argun river, 17 miles north-east of Manchouli). The first case from this spot (young Cossack) was diagnosed on September 17 at the Manchouli Hospital, when bacilli were seen in the smears from inguinal buboes. It is however almost certain that earlier infections had occurred in his family. The father had died in the middle of August at the

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(362) N. Manch. Pl. Pr. S. R. 1918-21.

(363) Plague in Wild Rodents.



Manchouli Hospital, the diagnosis being typhus. Two weeks afterwards two daughters succumbed. The young Cossack recovered in the hospital, while his mother died some days afterwards, apparently of *pneumonic* plague. The total is five cases.

- C. Kailastui (further north on the Argun river). Plague bacilli were found in smears from the axillary bubo of a Cossack arriving at Manchouli from this village and dying soon afterwards. His landlord in Kailastui had also died under suspicious circumstances.
- D. Kapzagaitui (on the Argun river). This interesting outbreak was apparently caused by 'free living' fleas. Dudshenko had already pointed that the fleas of wild Transbaikalian rodents emancipated themselves for prolonged periods from their hosts and led an independent existence in the steppe. It is quite probable that such insects played a role in the present outbreak. It was maintained that the infected hay harvesters did not hunt; on the other hand they had been so much bothered by fleas that they shifted camp. A Chinese party had hunted in the same region and had left a pile of skinned tarabagan corpses 2 miles away. In the Russian camp a youth as well as a girl fell sick. He developed an inguinal bubo but recovered. The girl died and infected members of her family. A chronological list is appended:

	<i>Date sick:</i>	<i>Result:</i>
Young Cossack .....	Aug. 2	Recovered.
Girl, aet. 13 .....	,, 2	Died on the fields.
Son, aet. 17 .....	,, 8	Died.
Father, aet. 50 .....	,, 15	,,
Girl, aet. 8 .....	,, 19	,,
Mother, aet. 50 .....	,, 21	,,
Total ..... 6 cases, mainly with inguinal buboes.		

This outbreak terminated in the second half of August. As stated in our Report 1918-22, the earliest suspected cases occurred at Hailar 117 miles *east* of Manchouli, at the beginning of October among some Chinese working in a fur factory. It was impossible to find out how and from where these became infected. Whether the infection was introduced into Hailar from the Transbaikalia foci or whether they originated in some focus nearer Hailar is not clear.

There is no need to give any further mention of the 1920-21 epidemic in the Chinese territory. The principal data

have been published in our previous Report (1918-22.) Our description of its progress in the Russian Coast Province (Vladivostok and district) is supplemented by a Report of the Soviet authorities <sup>(364)</sup>, a summary of which is given here.

A. General survey.

<i>Locality :</i>	<i>Patients :</i>	<i>Corpses :</i>	<i>Total :</i>
Vladivostok .....	209	289	498
Nikolsk-Ussurisk .....	10	11	21
Suchang Coal Mines ...	8	6	14
Coast Province . .....	—	17	17
	<hr/>	<hr/>	<hr/>
Total .....	227	323	550
	<hr/>	<hr/>	<hr/>

B. Course of epidemic in Vladivostok week by week (April 4-September 17) :

N.B.—The warm seasons embraced within these months should be noticed.

<i>Weeks :</i>	<i>Patients :</i>	<i>Corpses :</i>	<i>Remarks :</i>
1.	2	10	
2.	15	12	
3.	13	22	
4.	27	50	
5.	26	59	
6.	42	47	
7.	41	32	
8.	19	21	1 inguin. with sec. pn. recovd.
9.	13	7	
10.	0	3	
11.	0	4	
12.	0	1	
13.	0	2	
14.	2	2	1 cervic. fatal; 2 septic.
15.	4	0	2 inguin. 2 „
16.	0	2	2 „
17.	0	2	1 „ 1 „
18.	0	4	4 „
19.	1	2	1 pneumon.
20.	1	1	1 axill. „ 1 intestin.
21.	0	2	1 „ 1 „
22.	2	2	3 „ 1 „
23.	0	2	2 „
	<hr/>	<hr/>	
Total	208	289	

(364) Pn. Epid. in the Coast Prov. & Vladivostok 1921 (Sacharoff, Bjelajeff, Velmovski, Meibom). VI. 1922.



## Remarks upon Table B:

- i. During the first weeks, the majority of patients suffered from pneumonic plague. The report does not mention septicemic cases during that time, but it is possible that some, who died quickly in the 'isolation department', before a diagnosis could be made, suffered from septicemic plague. Within the first six weeks of the epidemic 26 such patients were counted, in three of whom pneumonic plague was diagnosed at p.m. Of the remaining 23, no definite type was entered, and therefore might represent the possible maximum of septicemic cases in a total of 151, thus putting the percentage of septicemic cases at 15.2%

The corpses dissected during the first weeks showed pneumonic plague in an "overwhelming majority". The report does not state how many *post mortems* were performed on patients succumbing in the hospital or how many on bodies found in the streets. Possibly the incidence of septicemia was somewhat higher among the latter.

Apart from this all evidence points to the fact that septicemic cases were not frequent in the early days of the outbreak, while their incidence was high towards the end. We observed a similar distribution in Harbin and wish to lay stress upon this confirmation of our findings. We pointed out in our previous report that those patients with septicemia are less infectious than those with pneumonic plague and we might see in the high incidence of such less infectious cases towards the end of the epidemic an explanation of the self limitation of pneumonic outbreaks. We emphasize again the importance of this aspect.

The authors of the Vladivostok report are inclined to connect this high incidence of septicemic plague at the end of the outbreak with a diminution in virulence of the invading organism. The warm weather might suggest such an explanation, but this has been often disproved by much epidemiological and experimental evidence.

- ii. The statistics given to show the incidence in different races are omitted, because, in our opinion, no racial immunity exists in pneumonic plague and any appearance of such may be explained by social condi-

tions. The Vladivostok report confirms this opinion when stating that whole classes of the Chinese population, e.g., the servants of Russian families, escaped the fate of their brethren who were exposed to infection in crowded quarters.

The mortality among the sanitary staff was as follows:—

1	sister	(working in wards and admitting patients)
2	feldchers	(working in isolation wards)
3	attendants	(2 working in plague wards, one in observation ward)
1	„	(working outdoors, coll. corpses and transport. pat.)
1	„	(working in pl. train, coll. corpses and transport. pat.)
<hr/>		
8	Total.	

### iii. Recoveries and healthy carriers.

We have mentioned already (Table B) that one patient recovered from bubonic plague and secondary pneumonia. This is the only case of recovery of confirmed plague at Vladivostok during the 1921 epidemic. The report lays some stress upon the fact that out of 184 patients admitted into the plague wards, 18 (9.7%) were discharged healthy. The authors take for granted that these had plague and apparently believe in a mild form in pneumonic plague, corresponding to *pestis minor* in bubonic plague. The history of only one instance is given in the report; the others are merely recorded as having entered the plague ward and come out alive!

The patient in question, a Chinese, was admitted on May 20. He complained of having headache and slight cough for two weeks. Plague bacilli were seen in his sputum on admission and on the 27th, samples bacilli were seen in his sputum on the 27th; samples seen on 28th were negative, while on the 29th 'suspicious' bacilli were found. Sputum was negative for B. P. on the 30th, when patient received 120 c.c. of anti-plague serum. The patient made an uneventful recovery, was sent on June 1 to the isolation wards and later on dismissed. No data about fever or lung



symptoms are given, and from the above it would seem rather probable that this man was a carrier and not one suffering from a hypothetical *pestis pneumonica minor*. A similar procedure might have been made in some other patients who left the plague hospital alive, but in view of our experience in Manchuria, these like the detailed case above should be classed under 'healthy carriers' rather than actual cases.

iv. Observations upon plague in rats.

The incidence of plague among rats in pneumonic outbreaks has been mentioned in former pages. For instance, Indian observers admit the possibility that pneumonic plague may pass from man to rat, and that the rat in its turn may cause bubonic plague in man. In the Astrakhan districts such results have not been obtained. During the first Manchurian outbreak (1910-11) no rat plague was found, though numbers of animals were investigated at several localities in North Manchuria and South Manchuria. In the 1921 outbreak no infected rats were observed throughout Manchuria.

In view of this negative evidence it is interesting to see that two infected rats were found in Vladivostok. They were delivered to the laboratory on June 24 and 25 respectively. Unluckily both specimens were unfit for macroscopical examination, so that the presence of buboes could not be determined. It seems, however, that one of the animals had possibly subacute changes as shown by nodes in the kidneys. Plague was confirmed by all tests. The report mentions the absence of any epizootic on a considerable scale among the rats, an assertion confirmed by no further invasions among the rat population at Vladivostok after the epidemic.

A summary is given below showing the *course of the 1921 epidemic in Transbaikalia*<sup>(365)</sup>.

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(365) Jetmar, Unpublished Russian paper.

Locality	Started	Ended	No. cases	Mode infection
i. Oppos Kailastui on Chin. territ. ....	Jan. 25	Feb. 20	? 5	Through pat. from Dalainor. — All pneumon.
ii. Opp. Kapzagaitui on Chin. territ. ....	Feb. 1	?	? 3	Through pat. from No. i. — All pneumonic.
iii. Kailastui .....	Jan. 27	Feb. 13	10	Through man inf. from No i. — All pneumon.
iv. Klichki (66 m. fr. Manchouli) .....	Feb. 13	„ 20	6	Thr. man inf. in Manchouli. — All pneumon. One patient comm. suicide showing at p.m. early stage of pneumonic pl. (366).
v. Substation 86.....	„ 28	March 4	2	1 pneumon., 1 bubon. (366).
vi. Mazievskaja (Quarant. st.).....	?	„ 9	3	Fr. Manchouli. All pneum.
vii. Chankyr .....	March 1	„ 1	1	Pneumon.
viii. Borzia .....	„ 3	„ 5	1	Discharged fr. Matsievskaja after 5 days quarant. Fell sick in train. Pneumonic.
Total			? 31	(30 pneumon., 1 bubon.)

*Discussion of bubonic cases in 1921.* In our Report 1918-22 we remarked upon the rarity of bubonic cases during the 1921 outbreak.

The occurrence of such bubonic cases during pneumonic outbreaks is interesting but difficult to explain. The presence of cervical buboes may be partly explained by an infection through the mucous membranes of the eye, nose, mouth, etc. Axillary and cubital buboes might be partly due to an entrance of infection through some wound in the fingers. In this respect a case reported from Transbaikalia<sup>(366)</sup> deserves mention: The wife of a railway-guard admitted a woman suffering from pneumonic plague into her house. When the patient began to soil the hut with her bloody sputum, she was evicted. The Plague Detachment found her dead near the railway and confirmed the diagnosis at p.m. Visiting the hut, they found the house-wife wiping away the sputum. Her fingers then showed scratches. Five days afterwards a bubo was discovered over right elbow. This suppurated on the sixth day, patient died of heart failure on the 9th. No lung symptoms were noted.

Regarding inguinal buboes, observed several times during the 1920-21 outbreak, no direct infection is possible in cold Manchuria. This is also not probable for Vladivostok, where people do not walk barefooted. The only conclusion is an infection through parasites. At Vladivostok there is

(366) Jetmar, Z. f. Hyg. & Inf.-Kr., 1923, p. 322.



a remote possibility that rat fleas played a role. On the other hand, we believe the role of ecto-parasites in man to be more important in such cases where no direct entry of the plague organism is possible.

1921: The pneumonic epidemic in Transbaikalia terminated in spring. The next autumn we announced the discovery of infected tarabagans by the Russian Plague Detachment. It was proved that an epizootic raged in the districts around Sektui (65 miles from Manchouli).

Several human outbreaks apparently independent of one another were observed in the second half of the year:

A. Dauria (38 miles from Manchouli, 30 miles fr. Sektui). The first two cases occurred in two railway employees (station master and pointsman) who had attended a hay-harvest 7 miles north-west of Dauria and had come in contact with tarabagans<sup>(367)</sup>. The first man developed a cervical bubo in the fields on Aug. 8 and was admitted into the military hospital at Borzia. He denied any excursion to the steppe and complained only of sore throat and dyspnoea. *Angina Ludovici* was diagnosed and an operation performed by Dr. Guruleff assisted by another doctor and a sister. They used no gloves. No pus was found and the patient died of suffocation three hours afterwards (Aug. 12). Dr. Guruleff performed the p.m. next day (with gloves) and, suspecting anthrax, took all precautions. The second victim (pointsman) reported sick on the 13th and died on the 17th showing cervical buboes. Dr. Guruleff himself felt unwell two days after the operation with cellulitis of the middle right finger where he had formerly seen a scratch. He developed a right axillary bubo and died on the evening of 17th. The next man affected with plague was an attendant Rogov, who had lent some assistance at the p.m. of the station master and had attended the doctor. He was found dead on Aug. 19, showing no buboes, only B.P. in blood. Both the assistant and the sister who helped Dr. Guruleff at the operation and a feldcher who took an active part in the p.m. with unprotected hands, escaped infection.

B. Haranor (16 miles w. of Dauria). A repair worker, after having skinned a tarabagan on Aug. 17, developed an axillary bubo on 19th and died on 25th<sup>(368)</sup>.

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(367) Krotkoff, Jl. Transbaik. Med. Soc., 1923, pp. 347-363.

C. Substation 83 (between Dauria and Haranor). A boy who had handled a dead tarabagan and from whom a tarabagan flea was picked up on Aug. 28, developed axillary bubo on Aug. 31 and died on Sept. 1.

D. Mulino (80 miles away from Railway). An ex-soldier having killed and skinned a sick tarabagan, developed an axillary bubo on Sept. 15 and he was followed by others, thus:—

	<i>Age :</i>	<i>Date sick :</i>	<i>Result :</i>
Brother .....	6	Sept. 24	D. Sept. 27
„ .....	7	„ 25	D. „ 27
„ .....	10	„ 26	D. „ 28
Sister .....	18	„ 26	D. „ 28
Father .....	45	Oct. 1	D. Oct. 4
Man (contact) .....	?	„ 1	D. „ 5
Brother .....	2½	„ 4	D. „ 6
Sister .....	12	„ 10	D. „ 12
„ .....	13	„ 13	Recovd.
<hr/>			
Total .....	10	(7 axillary, 3 inguinal bub.) <sup>(368)</sup>	

1922: A. At the end of July a suspicious case was reported in Kailastui.

B. At the end of September we were advised by telegram that 5 cases of pneumonic plague had occurred at Haranor. This information was alarming and, at the same time, surprising. For hitherto pneumonic cases have developed on a soil well prepared by preceding bubonic cases.

Personal investigations conducted by our staff (Drs. Chun and Kwan) revealed that the following cases had occurred in one family:

	<i>Age :</i>	<i>Sick :</i>	<i>Died :</i>
Girl	17	Begin of Sept.	After 9 days of 'pneumonia'
Father	47	? Sept. 12	? Sept. 15 of pneumon. pl.
Mother	44	? „ 18	„ 19 „ „ „
Sister	11	„ 18	„ 19 „ „ „
Brother	12	„ 18	„ 20 „ ? septicemic pl.
Total	5		

(368) L. c.; Sukneff, Publ. Harbin Med. School, No. I, pp. 215-234.



Owing to lack of education the inhabitants of Haranor were rather savage and on this occasion treated the Plague Staff violently.

The first patient was seen only by a feldcher who diagnosed pneumonia, though pain in the axilla was present. She refused any examination but there is reason to suspect that she had bubonic plague with secondary pneumonia. When the mother died, the villagers accused Dr. Krotkoff of having introduced infection into the family and locked him and 3 assistants in the house. The doctor had to stay in the same room with the dying girl (aet. 11). The three dressers attended the boy (aet. 12) in another room. The doctor, wearing no mask, nursed the dying girl until the end, but, when not occupied, stayed near the open window 5 feet away. Happily one dresser managed to escape and fetch a military doctor with an escort from Dauria who rescued the prisoners after 36 hrs. confinement. None of the staff was infected.

The outbreak was not originally of a pneumonic nature. This is proved by the fact that the first case had secondary pneumonia and also by some suspicious cases having occurred since August in the village. Amongst these, 5-7 children, from 10 months to 10 yrs. old, had died with a diagnosis of 'meningitis' by an inexperienced dresser. Further cases occurred at end of August in the fields in a peasant (aet. 47) and his daughter (aet. 17) again diagnosed as meningitis, as well as in a girl (aet. 8) who died at the same time as the first authentic case and showed similar symptoms.

C. A girl (aet. 17) smuggled tarabagan skins from Substation 76 into Manchouli. In order to hide them she made garments of them which she wore in direct contact with her skin. She fell sick on Sept. 19 and died after a week in the Oloviannaja Hospital with axillary bubo.

D. The last outbreak at Dauria may be summarised thus<sup>(369)</sup>.

	<i>Age :</i>	<i>Sick :</i>	<i>Bubo :</i>	<i>Result :</i>
Boy .....	10	Oct. 12	Inguin.	Recovd.
Brother ....	12	,, 22	,,	D. Oct. 25
Mother . ....	41	,, 23	,,	D. ,, 27
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Total .....	3			

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(369) Sukneff, Sib. Med. JI., 1923, Jan.-May; Georgievski, JI. Transbaik. Med. Soc., 1923, pp. 337-341.

- Note: i. Case No. 3 was 8 months pregnant. The foetus, dissected at p.m., had no macroscopical signs, but smears and cultures from spleen were positive.
- ii. The outbreak started two weeks after the hibernation period of the marmots. No. 2 was a keen hunter and continued till very late in the season. Many skins and much unboiled fat were found in the house. Both boys greased their boots with this fat. Sukneff<sup>(369)</sup> found on corpse of No. 2 15 *pediculi vestimentorum* in which plague bacilli were found and confirmed by cultures and experiments. Guinea pig experiments with live lice produced negative results.

1923: The outbreaks in this year are discussed in the paper on Wild Rodents and summarised thus:

- A. In Konzinor, near Jakoshe (168 miles east of Manchouli) a man, aet. 24, fell sick May 19, died on 22 with axillary bubo.
- B. At Haranor (52 miles west of Manchouli) a man, aet. 37, fell sick with axill. bubo May 16, died on 20.
- C. At station 83 (8 miles from Haranor) a girl, aet. 12, fell sick with axill. bubo on Sept. 1, died on 3.

#### *Summary of Transbaikalia:*

About 60 outbreaks have been known since 1863; in 50 of them we counted 271 cases of plague, whilst in about 10 their number is unknown.

Of these 271 cases were:

Not determined as to type ...	145	(among them 20 initial cases)
Undetermined bubonic .....	16	,, ,, 4 ,, ,,
Cervical buboes .....	10	,, ,, 2 ,, ,,
Axillary buboes ....	44	,, ,, 15 ,, ,,
Axill and inguin. buboes ..	2	,, ,, 1 ,, ,,
Axill and cervic. buboes ....	2	,, ,, 1 ,, ,,
Inguinal buboes .....	38	,, ,, 24 ,, ,,
Pneumonic .....	29	,, ,, 21 ,, ,,
Septicemic .....	5	,, ,, 2 ,, ,,
Total .....	271	,, ,, 50 ,, ,,

#### Note on the tabulation:

The above figures lack exactness in some respects. We attempted nevertheless to present such a summary because certain important conclusions may with safety be drawn:



- i. In the overwhelming majority of outbreaks, for which we have complete data, the initial cases were of the bubonic type. The preponderance of axillary buboes is well marked. This is in accord with the experience of McCoy in plague caused directly by the *Citellus*.

We believe that the axillary buboes of the initial cases are partly the result of a direct infection (skin-ning and cutting of sick animals) whilst the others are caused by the tarabagan flea. An infection through the eating of sick animals is highly improbable.

- ii. Later cases. The distribution of the buboes in the later cases, though perhaps not quite in accord with statistics from other countries, shows a considerable number of inguinal in addition to numerous axillary and rare cervical ones. It is our belief that those secondary cases are mainly infected through bites of human parasites which abound in the huts. The role of the domestic rodents and their parasites has thus far not been proved and seems improbable.

## 2. MONGOLIA.

There have been many outbreaks in Mongolia since 1860. The records, however, were not collected by a trained personnel but by occasional expeditions to the affected areas after the events and thus often lack essential details.

The conditions accompanying plague in Mongolia are in many respects similar to those of Transbaikalia. We find large tracts of the country populated by wild rodents (tarabagans, susliks, baibacs)<sup>(370)</sup>. The former are eagerly hunted, their skins being a kind of currency to buy tea and other commodities with from the travelling merchants<sup>(371)</sup>, and are undoubtedly responsible for the outbreaks. The measures taken against the pest by the population, lamas and authorities seem thorough. For instance, sick animals must be notified, and the spiritual heads (*Gegen*) prohibit hunting in the affected districts. If anybody disobeys, his horse and saddle are taken from him, otherwise he goes to jail<sup>(372)</sup>. Even special officials are said to be appointed in North Mongolia to supervise the enforcement of such laws<sup>(372)</sup>. As soon as disease breaks out, the neighbours avoid all contact with the sick and quickly shift their camp, especially in summer time. In winter this

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(370) Zabolotny, Arch. Russes de Path., 1899, pp. 242-250.

(371) Skchivan, ibidem, 1901, pp. 603-612.

(372) Wassilevski, Vj Guig., 1915, pp. 178-201.

measure is carried out only in case of extreme necessity because it involves great difficulties. It is realised that not only is touching the sick dangerous, but also their *breath* is infective. The lamas try to protect themselves by smearing an aromatic ointment round their mouth and nostrils. The affected families themselves try to evacuate the infected locality as soon as possible, and leave often their sick or dying in the huts while they stay in the open for '101 nights,' a period considered to be sufficient to remove all traces of danger. The dead bodies are rarely burned; the scarcity of wood makes this possible only in the case of a rich or important man (lama). Sometimes the whole *jurte* with the dead bodies is burned. Most often, however, the dead bodies are left on the open ground<sup>(373)</sup>.

It is related that in 1908 a contact, the sole survivor of an infected household, took refuge in a monastery, which as a result was shut up for 30 days. Nobody went in or out. Daily at a given hour all inhabitants assembled in the courtyard. Lamas of a neighbouring monastery climbed an adjacent hill and counted with the aid of their binoculars, if all were still healthy<sup>(373)</sup>.

Important factors concerning the limitation of plague outbreaks are the sparsity of the population (1907: 1,850,000 in a territory of over a square million miles) and the absence of railways and other means of quick transportation.

Plague is manifested not only in its bubonic form. Complication with secondary pneumonia leads to frequent pneumonic outbreaks.

The earliest tales about plague outbreaks reach back to the sixties of the 19th century<sup>(374)</sup>. Since 1886 almost yearly outbreaks have been recorded. In 1896—about the same time that plague became active in other parts of Asia—they began to assume comparatively large dimensions. The first medical observer, who reached the affected districts in the So-len-ko valley (Weichang district) was Matignon, physician of the French Legation in Peking. His observations<sup>(375)</sup> were supplemented and bacteriologically confirmed by Zabolotny<sup>(370)</sup>.

The 1896 epidemic began in July and lasted till November. At least 160 fatal cases were counted in ten villages; in some 13-15% of the population died. About one third of the patients showed purely pneumonic characteristics which prevailed mostly in the last 1½ months of the epidemic.

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(373) Dudshenko, *ibid.*, 1909, pp. 1045-1089.

(374) Talko-Grinzevich, *Przeglad Lekarski*, 1900, No. 15; *Transbaik. Gov. Rep.*, 1900, No. 7.

(375) *Ann. d'Hyg. Publ.*, Vol. 39, p. 227.



In 1897 a smaller epidemic was noted in summer.

In 1898 this Weichang district was visited by Zabolotny, as 24 cases had been reported there. He himself attended 16 more; of these 7 were pneumonic. Zabolotny states that in 5 villages with 658 inhabitants about 400 persons died during the years 1896-1898.

In 1899 only a few cases were observed by Damaskin who had travelled from Russia<sup>(376)</sup>.

In this year the pest was very active in other parts of Mongolia<sup>(374, 371)</sup>. Apart from a small outbreak in spring, affecting 3 tarabagan hunters in a camp 133 miles east of Kobdo (S. W. Mongolia) the following foci were noted:

- A. Three districts in N. W. Mongolia (266-330 miles from Urga). 200-300 cases in September.
- B. Kerulen River, N. E. Mongolia. 80 deaths.
- C. Two districts in S. W. Mongolia. No figures.
- D. Near Hailar in Mongolia. Two families died in autumn after eating diseased tarabagans.

Outbreaks A and B and probably C were mainly pneumonic in type. From 1900 till 1905 Mongolia seemed free from plague. This coincides with a quiet period in Transbaikalia, which, however, was more apparent than real. For instance, an expedition made by Schreiber<sup>(377)</sup> to Mongolia in 1905 arrived too late to find any patients, but reliable reports convinced him, that both epizootics and human cases were present in 1905 and *previously*. The human outbreaks (some bubonic) were limited.

In October 1908 the disease was imported from Kulusutai (Transbaikalia) into Mongolia, where with one exception all the relatives of the *Gegen* (spiritual head) died.

From this time onward no direct information is forthcoming about outbreaks in Mongolia. A Russian doctor mentions<sup>(378)</sup> a prevalence in the Ordos country and other parts of Inner Mongolia in August 1917 and this is confirmed by Chinese reports, that a 'winter sickness' occurs periodically in those parts. Preshevalski<sup>(379)</sup> speaks of the presence of tarabagans in the Ordos country.

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(376) Kersakov, Vj. Guig., 1900, p. 676.

(377) Russki Vrach, 1907, pp. 289-292.

(378) China Med. Jl., 1918, pp. 146-147.

(379) Qu. fr. Podbjelski, Arch. R. de Path., 1901, p. 254.

In 1917 plague was active in the Patsebolong district of South Mongolia. These parts were perhaps the main distributing centres for the 1917-18 *Shansi epidemic*, which may now be discussed<sup>(380)</sup>.

### SHANSI EPIDEMIC.

As far as can be ascertained, this epidemic started from the Patsebolong district. It is true that about the same time a limited outbreak, killing 50 persons, was reported at Mai-Uh in Thibet followed by 25 more in Oct. in the Moslem Community of Taochow (Old City) in Kansu Prov. These parts are too far from the Patsebolong zone to have any bearing upon the Shansi outbreak.

From Patsebolong it travelled eastwards to Paot'ouchen and Saratsi, then invaded Suiyuan and Kweihua, from which it spread later on eastwards to Fengchen, Tatungfu and the railways, and southwards to Soping, Tsoyun, Sochow, through the Great Wall into Taichow, Hsinchow, and neighbourhood of the capital city of Taiyuanfu. Isolated cases also occurred afterwards in Hsuanhuafu, Peking, Tsinanfu, Pengpu, Chengting, and even Nanking on the South of the Yang-tse River.

Altogether a vast area of ground was covered, comprising eight provinces (Southern Mongolia, Suiyuan, Chahar, Shansi, Chihli, Shantung, Anhui, Kiangsu) and five railway-lines (Peking-Suiyuan, Chengting-Taiyuan, Peking-Mukden, Peking-Hankow, and Tientsin-Pukow).

It is interesting to note that in this, as in the great Manchurian Plague of 1910-11, the epidemic started in the waste regions of the North towards the end of autumn and disappeared at the approach of spring. The last case was reported on March 20, the total number of victims being about 16,000. The distance affected from seat of origin to terminating point was 1600 miles. The epidemic remained comparatively limited, mainly on account of the fact that railway areas were involved only secondarily, when measures had already been taken to control its spread. Nevertheless the pest was able to perform some "remarkable long distance sprints" (Stanley), once the railway system was reached, reaching for instance Nanking, which city was attacked on March 8; but here only

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(380) Off. Rep., Min. of Home Aff., Nat. Med. Jl., 1918, p. 88.

Young, Rep. of Shansi Pl. Prev. Bureau, Peking 1918.

Stanley, Nat. Med. Jl. 1918, pp. 42-44.

Parry, Ch. Med. Jl., 1918, pp. 81-87.

Roys, *ibid.*, pp. 346-348.



20 cases developed. The spread in the earlier part of the epidemic was "limited to a day's travel by animals; usually about 20-30 miles a day" (Young).

We have no record of any bubonic case in this epidemic but the septicemic type occurred (Young). One possible case of recovery was reported by Ingram in a severely affected family. The patient (female) showed plague bacilli in her sputum. Her husband was later attacked and died, but she recovered.

The following statistics of sexes for two districts are supplied by Young:

	<i>Deaths m.</i>	<i>Deaths f.</i>	<i>Sex ?</i>	<i>Total</i>	<i>m.</i>	<i>f.</i>
i	530	256	197	983	67.4	32.6
ii	501	271	526	1398	68.9	31.1
Tot.	1131	527	723	2381	68.2	31.8

It is evident that the Manchurian epidemic of 1910-11 was principally an epidemic in a "floating population" and thus more liable to kill the adult males, while the Shansi epidemic affected the domestic homes more.

The mortality among the sanitary staff was low, as only 8 native practitioners and two burial coolies were reported dead.

Pneumonic plague was again reported from Shansi (Linhsien) in December 1918 and Jan. 1919, but the diagnosis was not confirmed bacteriologically<sup>(381)</sup>. The 91 cases noted were all fatal. Naturally the question arises, whether this might not have been the fatal pandemic of influenza which was invading China as well as other countries at the time.

In late summer and autumn of 1919 the existence of bubonic plague, principally inguinal, was proved microscopically at Linhsien. 350 deaths occurred, only five percent recovering. The same disease had apparently occurred in a mild form in the district during two previous summers. The etiology of these outbreaks remains obscure, as few of the villagers had seen dead rats. No rodents could be procured for examination<sup>(382)</sup>.

With reference to *Thibet* no definite information is available as to the type of the disease present in this isolated country.

(381) Watson, Ch. Med. Jl., 1919, pp. 169-173.

(382) Watson, Nat. Med. Jl., 1920, pp. 93-97.

## REMARKS UPON TRANSBAIKALIA AND MONGOLIA.

We have stated repeatedly that the outbreaks recorded in the foregoing pages are not complete. Still, with the facts at our disposal we can say that an epidemical wave—as it were—could be detected rising and falling almost regularly in these regions. Thus, extensive visitations are noted in the following years :—

Year	Location	Remarks
1896-99	Mongolia .....	Considerable pneumon.
1905	„ and Transb. ....	None pneumonic.
1910	Transb. and Manchuria .....	Big pneumonic.
1917	Mongolia and Thibet.....	„ „
1920	Transbaikalia and Manchuria	„ „

It is difficult to fathom the mystery of this apparent periodicity, resulting in a greater activity of plague almost every fifth year and leading in four of five instances to widespread pneumonic outbreaks. An explanation may be found perhaps in the tides of prevailing epizootics. In view of our lack of knowledge regarding the character of such epizootics, it seems hazardous to draw any definite conclusions. But it may be pertinent to ask whether such as we have noted above are due to a contiguous spread of infection among the rodents or to their migration caused by some extraordinary event, such as excessive hunting<sup>(383)</sup> or scarcity of food due to parching heat. In this connection it may be pointed out that the year 1905 was an unusually dry one<sup>(384)</sup>. Further elucidation of this problem seems advisable.

## XII. PNEUMONIC PLAGUE ON SEA-VESSELS AND IN LABORATORIES.

### 1. PNEUMONIC CASES AND OUTBREAKS ON BOARD SHIP.

We possess some notes of apparently pneumonic cases on vessels in historical times, but nothing can be gained by their discussion. We therefore concentrate upon the present pandemic. The following instances are on record.

(383) Radde, quoted by Skchivan. l. c.

(384) Rjeshetnikoff, Vrach. Gaz., 1908, pp. 207-208.



Year	Vessel	Bound from	To	No. c.	Etiology; remarks
1896	?	Calcutta	London	1	? (385)
1899	Centauro .....	Montevideo	Asuncion	2	Susp. Rice bags fr. India (386)
1899	Mersey .....	Calcutta	Demerara	9	„ Among coolies (387)
1899	Haidari .....	Djeddah	Bassorah	? 1	„ „ pilgrims (388)
1899	American Maru	The West	Honolulu	1	„ ? (389)
1901	Friary .....	Alexandria	Hull	8	
1899	Mirzapur .....	The East	Camaran	6	„ „ pilgrims (390)
1904	Korea .....	Hongkong	Kobe	1	Rats unaffected. (391)
1905	Aida .....	Suez	Port Said	2	First c. app. inf. on sh. (392)
1906	Indus .....	Calcutta	Trinidad	7	Susp. Among emigrants (393)
1906	Calypso .....	Venice	Trieste	1	Jute and cotton fr. Ind. (394)
1907	Wharfedale .....	Buen. Ayres	Hamburg	1	Susp. Rats app. inf. (394)
1910	Bohemia .....	Bombay	Aden	1	? (395)
1912	Zafiro .....	Hongkong	Manila	1	Board. in incub. stage? (396)
1912	Cheongshing ...	Hongkong	Tientsin	1	Purser's wife inf. Hkg. M. P. P. S. Report 1911-13.
1912	Loongsang .....	„	„	1	Board. in incub. stage? (396)
1919	Nagoya .....	The East	London	8	Prec. app. by bub. c. (397)
1920	Alps Maru .....	Japan	„	? 1	Prec. by bubon. c. (398)
1921	Kisheneff .....	Vladivostok	Chefoo	12	Inf. br. aboard in VI. (399)
1921	Ralph Moeller ...	„	„	3	Inf. br. aboard in VI. (400)
1922	Polycarp .....	Ceara	Para	1	Board in incub. st.? (401)
1922	City of Genoa ...	Bombay	London	1	Prec. by bub. cases (402)

Most of these instances present no unusual features, the pneumonic cases being due either to preceding bubonic ones or to infection received on shore.

There are, however, two instances which might possibly be termed 'original' cases of pneumonic plague.

(385) L. G. B. R. 1896-97.

(386) Ibid., 1898-01, p. 429.

(387) „ p. 425.

(388) „ p. 163.

(389) „ p. 365, 385.

(390) „ p. 156.

(391) „ 1904-05, p. 254.

(392) „ 1905-06, p. 366.

(393) „ 1906-07, p. 98.

(394) „ 1907-08, p. 250.

(395) „ 1910-11, p. 162.

(396) Heiser, Saigon Conf. Rep. (1913), p. 228.

(397) Min. of Health Rep., 1919-20, pp. 203-205.

(398) Ibid., p. 210.

(399) Vladivostok Rep., pp. 37-43.

(400) N. Manch. Pl. Pr. S. Rep. 1918-21.

(401) U. S. P. H. R., 1923, Sept. 21, p. 2210.

(402) B. M. Jl., 1923, May, pp. 896-897.

The previous pages have shown that such instances supposed to arise *de novo*, if existent at all, are extremely rare. In areas where repeated outbreaks have been studied, like Egypt and Transbaikalia, their existence is denied. Some cases were said to occur in Astrakhan, but we find that on every occasion their true nature was only surmised, not properly diagnosed. Only a few similar instances from other parts of the world (see outbreak in Algeria, 1912) are on record which may deserve the benefit of the doubt, but even these are not absolutely confirmed. It is our present task to investigate the two outbreaks arising in the confined space of vessels and others arising under laboratory conditions.

- A. *S. S. Friary*: This ship, with a crew of 21 persons, left the port Alexandria on Dec. 22, 1900, after a stay of 12 days; the whole crew had been on shore during this time. The vessel, carrying cotton seed from Alexandria to Hull, was in Algiers on Dec. 30-31, arrived in Hull on Jan. 10, 1901. Some dead rats had been found on her after sailing from the Egyptian port. 12 hours before the ship arrived in Hull, one sailor died of 'influenza.' On Jan. 12 two sailors fell sick with lung symptoms, diagnosed afterwards as pneumonic plague. Up to the 21st 8-9 persons, including a private practitioner, were attacked. All died with exception of the doctor, who recovered, having shown but few plague bacilli in his sputum<sup>(403)</sup>.

The first patient had not been seen medically, and it is therefore impossible to state what type of plague he suffered from.

A cat came aboard at Alexandria and was found ill after arrival in Hull. Unluckily it was killed by one of the crew and thrown overboard<sup>(404)</sup>, so that the important question of the nature and type of its infection remained unsettled.

- B. *S. S. Calypso*: This steamer plied between the two healthy ports of Venice and Trieste. Towards the end of October 1906 she took into Trieste a cargo of cotton and jute which had been brought to Venice from India by another steamer. A quarter-master who had to do with this trans-shipping fell sick and died at Trieste on Nov. 8. The *post mortem* roused a suspicion of pneumonic plague, which bacteriolo-

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(403) Reece, Rep. Min. of Health. 1919-20. pp. 284-285.

(404) L.G.B.R. 1898-01, pp. 14-16; 1901-02, p. 332.



gical examination confirmed. 2 rats and 18 mice, found on board after fumigation, were free from infection. This case, fully confirmed, may be regarded as a true 'original' pneumonic one<sup>(405)</sup>.

## 2. LABORATORY OUTBREAKS.

Chronological survey:

Year	Locality	No. cases	Remarks
1898	Vienna .....	1 + 2	
1901	Ann Arbor .....	1	
1903	Berlin.....	1 + 1	
1904	Petrograd .....	1 + 1?	
1907	" .....	1 + 1?	
1907	Lima (Peru).....	1 (fatal)	Accid. infect. with pl. cult. (406). Type?
1909	London .....	1	
1910	Odessa .....	1	Student, prob. infect. in lab. (407). Type?
1911	Harbin .....	1	
1915	Java .....	1	
1917	New Caledonia...	1 (fatal)	No details (408). Type?
1923	Chita .....	1 + 1	

Note: + indicates secondary cases resulting.

A. *Vienna.* The plague research work undertaken by the Austrian Plague Commission after their return home from India was practically finished when their attendant became infected. There was only one cutaneously infected guinea-pig in the stable, inoculated Oct. 4. On Oct. 14 the attendant Barisch, who looked after the animals, began to sicken. His case seems to have been rather atypical, because Mueller, the clinician of the Commission, declared that until a few hours before the death of the attendant the latter was not suffering from plague. Another possibility is, however, that Mueller realised the seriousness of Barisch's disease but kept it secret in order to protect his colleagues on whom some blame might fall. This is likely, because Mueller's publications show that he made light of the infectiousness of plague even in its pneumonic form. If so, his tragic error was to compare Indian conditions, where he observed the patients under a warm climate and in half open wards,

(405) Ibid., 1906-07, pp. 106-107: Wr. Klin. Woch, 1907, No. 7.

(406) L. G. B. R. 1907-08, p. 239.

(407) Ibid., 1910-11, p. 166.

(408) Ibid., 1914-17, p. 94.

with conditions prevailing in Europe in autumn. The attendant infected himself most probably through carelessness on his part; he unknown to his employers — was leading a somewhat dissipated life and probably under the influence of liquor. Two innocent lives were sacrificed through his carelessness. On Oct. 20 one of Barisch's nurses fell sick; she received repeatedly antiplague serum and showed several times temporary improvement but she succumbed on Oct. 24. Dr. Mueller fell sick on Oct. 21 and died on 23rd. He had refused serum treatment.

The diagnosis of pneumonic plague was bacteriologically confirmed in all three instances. No post mortem was performed<sup>(409)</sup>.

- B. *Ann Arbor*: In April 1901 a student working with plague cultures sent from San Francisco to the laboratory at Ann Arbor (Michigan, U.S.A.) fell sick with symptoms suspicious of plague<sup>(410)</sup>. He was immediately treated with serum<sup>(411)</sup>. The temperature soon fell to normal and patient recovered.

Our information about this instance was somewhat scanty so that we were not quite certain of its real nature. Dr. McCoy, Director of the U. S. P. Health Lab., informed us, that this case "is said to have been pneumonic plague." This instance of laboratory infection seems doubtful.

- C. *Berlin*: Dr. Sachs, a young Austrian doctor, working in the "Infectious Diseases Institute" accidentally infected himself May 28, 1903, while puncturing the bubo of a guinea-pig and then spraying the contents of the syringe upon an agar dish. It was thus assumed that infected droplets invaded the mucosa of his mouth or nose. He fell sick in the night between June 2 and 3 and developed slight tonsillitis, oedematous swelling on the right side of the neck and pneumonia. After his death of June 5 a partial *post mortem* was performed. The neck was not dissected and the presence of a cervical bubo could not be ascertained.

The contacts received prophylactic serum (45 c.c.). One, a male nurse Markgraf, fell sick with fever and showed after three days bloody sputum. The presence

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(409) Rep. of Austrian Pl. Comm., Vol. I, pp. 227-228, III. pp. 583-589.

(410) L. G. B. R. 1901-02, p. 302.

(411) Hewlett, Serum & Vaccine Th., 1910, p. 214.



of B. P. was proved by all tests. He made a good recovery under serum treatment, being given doses from 450-600 c.c. <sup>(412)</sup>.

While the case of the attendant is one of the best confirmed instances of recovery from primary plague pneumonia, we are not sure that Dr. Sachs suffered from this form.

- D. *Petrograd*. Both infections occurred in the plague laboratory established in an old fort on one of the islands of the Kronstadt Bay. Prof. Vishnikevich, the victim in 1904, had made inhalation experiments from Dec. 28 to 31 and had taken part in the preparation of plague toxin when plague bacilli, frozen with liquid air, were triturated. It is presumed that he became infected during one of these tasks. He fell sick with marked rigor and vomiting on Jan. 3, 1904. He developed a pneumonic focus at the angle of the right scapula. Sputum contained first B. P. mixed with diplococci and streptococci; on Jan. 5. plague bacilli were found in pure culture. Serum was administered (Jan. 5—100 c.c. subcut.; Jan. 6—200 c.c., partly subcut., partly intrapleural; then three times 100 c.c. intravenously). He died on Jan. 7. He had made his own diagnosis and adjured his colleagues to continue plague research.

At p.m. plague pneumonia was found in the upper and lower lobes of the right lung; the left lung, which showed old pleuritic adhesions, was free<sup>(413)</sup>. The peribronchia and deep cervical glands were much enlarged. "The enlargement of the deep cervical glands," concludes Zabolotny<sup>(414)</sup>, "and the abundance of bacilli in them shows, that primary infection occurred through the mucosa of the mouth."

A feldcher, attending Vishnikevich, fell sick on Jan. 6 with headache and chill. He developed fever; vocal fremitus over left lung was more marked and fine moist rales were heard over this lung. He received altogether 400 c.c. of serum and recovered after 5 days. No B. P. were found in smears of sputum and blood, but a positive agglutination 1:100 during his convalescence raised suspicion that he suffered

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(412) Doenitz, Berl. Kl. Woch., 1903, p. 609; Mukden Rep., p. 134.

(413) Mukden Rep., p. 160.

(414) Pestis Bubon., pp. 89-92.

from plague<sup>(415)</sup>. Simpson<sup>(416)</sup> states that two other attendants died of plague, but no confirmation is found in our Russian sources.

Dr. Schreiber fell sick with pneumonic plague on Feb. 14, 1907 and died on 17th<sup>(417)</sup>. Administrations of more than 1000 c.c. of serum were without avail<sup>(418)</sup>. Shurupoff<sup>(419)</sup> states that in this case also the infection occurred through the mucous membranes and that infection spread to the lungs through the lymph vessels. Dr. Padlevski infected himself at the p.m. of Schreiber and contracted bubonic plague. He recovered under serum treatment.

E. *London*: Dr. Parkinson, "an enthusiastic and most promising investigator," connected with the Indian Plague Commission, became infected while working with plague cultures in the Lister Institute (England). He fell sick on Feb. 1, 1909 with what was first thought to be influenza. The diagnosis of pneumonic plague was, however, made soon afterwards and he died on Feb. 4<sup>(420)</sup>.

F. *Harbin*. Dr. Michel, one of the doctors on duty at the Russian Plague Hospital in Harbin in 1911 epidemic, felt unwell on the evening of Jan. 18. He first paid little attention to his condition, even when bloody streaks could be seen in his sputum, because he had been suffering from a chronic lung affection. He then made a microscopical examination of his sputum and detected B. P. Patient died of pneumonic plague on the 22nd<sup>(421)</sup>.

Though it is probable that he contracted the disease in the wards, he believed he was infected in the laboratory while preparing smears from plague sputum. (He was in the habit of carrying samples of sputum and cultures in his coat pocket.—ED.)

G. *Java*. Borger, the Deputy Director of the Government Vaccine Institute at Weltevreden died of plague in November 1915. This was the second time he was infected with the disease. Some years before he died, a flask containing living plague bacilli broke in his hand, the liquid being spilt over the wounds caused

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(415) Rep. Russian Pl. Comm., II., p. 125.

(416) P. 79.

(417) R. Vr., 1907. p. 287.

(418) Mukden Rep., p. 102.

(419) R. Vrach, 1911, pp. 1097-1106.

(420) Jl. of Hyg., Vol. X. Pl. Suppl., p. 536; L. G. B. R. 1909-10, p. 59.

(421) Chmara-Barshevski's Rep., p. 376-377.



by the broken glass. His wounds were cauterised and plague serum was applied repeatedly with best results. His infection in 1915 remained undiagnosed; it occurred while he was bent upon preparing a new plague vaccine, working with broth cultures. He died after an illness of four days on Nov. 30. Diagnosis was fully established at p.m.<sup>(422)</sup>.

- H. *Chita*. The first victim was a lady doctor, Spielberg, who was a voluntary part-time worker in the Plague Department. Though she had some bacteriological training and had passed through the 1910 epidemic in Harbin, she was prohibited from messing with living plague bacilli. She disobeyed this order during the temporary absence of the chief. While preparing plague vaccine she spilt some of the emulsion on her gown. This occurred between Dec. 8-10, 1923. On 13th she fell sick complaining of pains in the chest. Some fever appeared, but the patient, trying to hide the seriousness of her condition, denied the possibility of any infection. Hence it was not until Dec. 15, that the diagnosis was established by the presence of bloody sputum. This showed B. P. microscopically and culturally besides streptococci P. M. on Dec. 16 showed confluent lobular pneumonia in both lungs. The extreme cold and other untoward circumstances allowed only a partial p.m.

Several persons had been in contact with the patient. One feldcher Michurin, aet. 31, became infected. He had attended her during the night from the 14th to 15th, staying as often as possible in the room next to hers. Early on the 15th he gave a new compress to the patient, without a mask, and it is believed he was infected at this time. He fell sick on the 18th and died of pneumonic plague on the 20th.

NOTE:—It can be seen from the foregoing pages that not all of the laboratory infections, diagnosed as pneumonic plague, were of a primary character. It is difficult to decide this question in the two Petrograd cases. The statements of Zabolotny and Shurupoff might suggest a secondary pneumonia, but it must not be forgotten, that many Russian authors consider all pneumonic infections to be contract-

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(422) L. G. B. R. 1914-1917, p. 73; Meded. van den Burg. Geneesk. Dienst in Ned. Indie, 1916, Part V, 1, pp. 1-4.

ed through the mucous membranes of the mouth or upper respiratory tracts and are thus inclined to consider changes found in the cervical glands as primary. For this reason it is difficult to interpret the p.m. findings of the Petrograd cases. However, cases like those in Java and on board the "Calypso" may certainly be classed under 'primary plague pneumonia.'

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### XIII. GENERAL CONCLUSIONS.

We have reached the end of our long compilation.

Only a few conclusions may be drawn from such historical records as we have assembled in the foregoing pages.

Some former observers, when trying to explain the problem of pneumonic outbreaks, were inclined to lay most stress upon meteorological influences. We do not deny such influences. For this reason, we have carefully recorded, wherever possible, the presence of cold, rainy seasons, etc. But it would not be accurate to regard such unfavourable factors as always present and solely responsible for the pneumonic outbreaks. It is perhaps permissible to say that these meteorological conditions form only one link in a chain of *extrinsic* factors which favour the *spread* of pneumonic plague. It seems to us an open question whether these extrinsic factors alone might explain the *rise* of pneumonic outbreaks, due to the ever present secondary lung complications, or whether we have to count with *intrinsic* factors as well. Such intrinsic features may include the character of the rodents and fleas involved, the changing nature of the plague organism under *varying* circumstances, etc. It is our belief that these latter factors help to mould the character of epidemics and to explain why pneumonic plague appears almost regularly in certain districts and infrequently in others.

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**PLAGUE IN WILD RODENTS INCLUDING LATEST  
INVESTIGATIONS INTO THE ROLE PLAYED BY THE  
TARABAGAN.**

(WITH 7 ILLUSTRATIONS.)

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*(Read at the Far Eastern Congress of Tropical Medicine,  
Singapore, September 1923.)*

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I. INTRODUCTION.

It may be remembered that while referring to the role which the tarabagan plays in the epidemiology of plague in our Service Reports 1918-1922<sup>(1)</sup> we said that only a few cases of natural plague had up to then been found among these free living animals.

Since that paper was written a series of epizootics, more or less severe, have been encountered by us in the spring of 1923 simultaneously in two separate regions, namely:—

- a. The old endemic area of Transbaikalia, to which frequent attention had been drawn in the past.

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(1) Br. Jl. of Hygiene, May 1923.

- b. A limited focus situated in former Mongolian territory, but now included as part of North Manchuria, lying north of the Chinese Eastern Railway in the neighborhood of the small station Yakoshih (between Hailar and Mentuho). This is the second occasion on which plague had originated in that region, the previous outbreak being in 1905.

For some time we have been collecting the necessary material for a comprehensive history of plague in Transbaikalia and Mongolia and take this opportunity of making a critical survey of the tarabagan problem up to date. Incidentally, a resume of the relationship of the order Rodentia (except *Mus rattus*, *Mus norvegicus* and *Mus musculus*) to plague is given. A list of rodents susceptible to either natural or experimental plague is also supplied. Although perhaps not quite complete, it will be found to be the most exhaustive that has hitherto been furnished.

(See Table I.)

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## II. WILD RODENTS SUSCEPTIBLE TO NATURAL INFECTION.

1. *Tarabagan*. As this stands by itself in importance a separate chapter will be devoted to it.
- 2—4 (*Kirghiz regions*.)

A list of the rodents in the Kirghiz steppes is given by Klotznitzki<sup>(2)</sup>. This contains only a few scientific names and is as follows:—

1. *Susliks*.
  - a. "Sand" suslik, called Large variety (600 grm.) Lives "Baibak" by the in southern part of steppe. Kirghese Kl. considers it as main reservoir of plague. Hibernates. Hunted for its fur. (*spermophilus rufescens* of his second paper).
  - b. Ordinary suslik Small variety (60-190 grm.). (*spermophilus musicus*) Lives in steppe containing more solid ground and in Kalmuck st. Hibernates. Destroyed because dangerous for crops. Found naturally infected.

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(2) Vj. Obst. Guig. 1912, p. 323 & foll.; Russki Vrach, 1913, No. 30, p. 1069.



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|----|---|---|
| 2. | Tushkaushik-jerboa<br>(at least two varieties)    | Most probably hibernates.<br>Holes not deep, easily hunted.<br>Found naturally infected.                          |
| 3. | Hare<br>(2 varieties: common and<br>"earth" hare) | Hunted and kept in captivity.   |
| 4. | Steppe mouse                                      | Non hibernating. Migrates in<br>winter to settlements and<br>penetrates into houses.<br>Found naturally infected. |
| 5. | Rats  | Found only round big settle-<br>ments with stores. Usually<br>unconnected with plague.                            |
| 6. | Hamster   | Experimentally susceptible.   |

*a. Experimental Evidence:*

Of these the following were found experimentally susceptible: Tartakovski<sup>(3)</sup>: the jerboas (most susceptible), susliks and field mice (less susceptible) and hamster (least susceptible). No marked macroscopical changes were found in the jerboas and susliks. Konstanzoff<sup>(4)</sup> used susliks for experimental purposes and found them susceptible. The animals showed moderate local reaction, no marked buboes, little localisation, death being due to septicemia. Shurupoff<sup>(5)</sup> experimented upon a large scale with the *spermophilus guttatus* from the Ural Govt. He recorded positive results from infection by cutaneous, subcutaneous, feeding, nasal, conjunctival methods and by contact. He noted particularly that contacts of those infected by the nostrils also died of the disease. Klodnitzki<sup>(2)</sup> infected susliks, baihaks and jerboas subcutaneously and found the small jerboas to be the most susceptible, then the baihaks, the susliks and big jerboas least susceptible. A paragraph in the Lancet<sup>(6)</sup> also stated, "it has been shown by experiment that infection of this animal (suslik) may be greatly prolonged, especially during the hibernating season."

Experimental evidence was supplemented by Tshurilina and Nosina<sup>(7)</sup> who immunised susliks with vaccine against subcutaneous infection with plague, and observed prolongation of the disease in vaccinated animals inhaled afterwards with

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(3) Vrach 1900, No. 33.

(4) Vj. Obst. Guig. 1903, pp. 1282-1307.

(5) Zentralbl. f. Bakt., 1912, vol. 65, No. 4-5, pp. 243-256.

(6) Nov. 1913, p. 1334.

(7) Russki Vrach, 1914, 10.

*bacillus pestis*. Tshurilina<sup>(8)</sup> found no B.P. in 4 susliks succumbed to plague and buried afterwards for 5 weeks in wooden boxes.

*b. Natural plague in the Astrakhan rodents:*

The rodents in these parts other than the tarabagan have in the past attracted insufficient attention. While the inhabitants of Transbaikalia and Mongolia have dreaded the tarabagan since immemorial times, the Kirghese did not seem to have realised the danger from wild rodents. This may be due partly to their shallow knowledge regarding plague. Furthermore the Astrakhan rodents were not hunted on such a large scale as the tarabagan for their fur. A curious fact lies in the lower incidence of plague in spring when the hunting season is at its height. The small susliks are a real pest to the crops and are for this reason destroyed in spring by women and young persons. They are also hunted for the sake of sport by children. Koltzov<sup>(9)</sup> mentioned a possible means of contact between man and susliks while collecting a plant (*agrifolium arenarium*) for food. In some instances infection was probably effected by fleas of the rodents jumping on persons working or staying over night in the open.

One very interesting and strange fact is reported by Palmurski<sup>(10)</sup> who asserts that the Kirghese know and fear plague and take measures against it. One of these measures consists in eating of susliks as a prophylactic. This went so far that at the time of the Vetlianka epidemic rich Kalmucks paid up to 20 roubles for one animal.

The earliest observation of an epizootic in these districts by medical men was perhaps that mentioned in Simpson's book<sup>(11)</sup>: "Dr. J. F. Payne informs me that in the plague on the Volga in 1878 and 1879, which he and Dr. Colville investigated, a large mortality among rodents was observed, but its relationship to the epidemic of plague did not impress him at the time. Now he is inclined to think that the association was very intimate." Only an outline of the positive and negative evidence regarding epizootics in the Astrakhan steppes can be given here. For a long time insufficient attention has been given to wild rodents as a prime cause of plague in these regions, especially in view of the discouraging results obtained by such observers as Tarskovski<sup>(3)</sup> who examined over 4000

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(8) JI. f. Microbiology. 1914, 1-2.

(9) Bull. de l'Office Internat. d'Hyg., July 1912.

(10) Rep. Astrakhan Med. Soc. 1909, No. 2, p. 7.

(11) p. 99.



animals, and Shukevich and Dominski (quoted by Klodnitzki<sup>(2)</sup>) who examined 3000 rodents, all negative. Nevertheless it cannot now be disputed that the freeliving rodents are really responsible for successive epidemics. Deminski<sup>(12)</sup> and Berdnikow<sup>(13)</sup> actually noted plague in susliks (small variety) and jerboas caught in the fields. They also reported cases where the smears showed suspicious bacilli, though neither cultures nor experiments proved positive. This observation coincides with our results respecting tarabagan carriers<sup>(1)</sup>. Deminski and his pupil Krassilnikova who attended him both died of pneumonic plague in the course of their research work<sup>(14)</sup>.

As already mentioned Klodnitzki<sup>(2)</sup> was inclined on epidemiological grounds to consider the *spermophilus rufescens* as the main reservoir and to regard the infection found in the small variety as one of "an episodical character." But he stated expressly, that they can also infect human beings, as is shown in the case of Deminski.

In the years following these investigations, big epizootics were observed among the susliks and also among the wild mice<sup>(15)</sup>. The latter which do not hibernate but migrate at beginning of cold weather to the villages and even invade the houses, are held responsible for the winter outbreaks. Nikanoroff<sup>(16)</sup> found that the domestic mice and *Mus norvegicus* were affected besides the wild mice in an outbreak in the Ural Gouvernement in 1915. Researches upon the fleas of the suslik have been carried out during a scientific expedition under Zabolotny<sup>(17)</sup>. The suslik fleas were found to attack man. Attempts have been made on a large scale to destroy the rodents, but evidently without success<sup>(18)</sup>.

5. (*Semiretchinsk*). According to the Russian Health Report<sup>(19)</sup> an outbreak of pneumonic plague in the Atbashinsk district, on the Aksai plateau, was traced to a "black marmot" caught and skinned by the first victim. Though this piece of information is scanty, we wish to lay stress upon it as confirming our belief in the existence of plague epizootics in these regions.

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(12) quoted from Klodnitzki, Russki Vrach. 1913, No. 30, p. 1069.

(13) Zentralbl. f. Bakt. 1913, Vol. 69, pp. 251-259.

(14) Trop. Dis. Bull. I/p. 549.

(15) Zabolotny, Archiv f. Schiffs & Tropenhyg., 1922, 12, p. 382.

(16) Rev. Microbiol. et Epidemiol. Saratov. 1922, p. 71-72.

(17) Annales Inst. Pasteur, June, 1923, p. 622.

(18) Lancet 1913/II. p. 1576.

(19) Otchet Nar. Zdravitsa, 1907, p. 162.

6-8 (*North America*).6. *Californian Ground Squirrel*.

This animal is interesting for many reasons:

Plague in North America is of comparatively recent date. McCoy<sup>(20)</sup> said that "the first cases which ever occurred in North America were recognised in San Francisco in 1900, the infection having come from Honolulu where cases had been carried for some months, or from a more distant Pacific port."

A study of the outbreaks in the first years showed that these had nothing to do with the squirrels. This is expressly stated in Bruce Low's Report for 1908<sup>(21)</sup>. "It is surmised that rats suffering from plague infected the ground squirrels in the first instance." McCoy,<sup>(22)</sup> to whom the investigation and eradication of plague in the Pacific states of America are mainly due, gives the following historical survey of the problem: "According to Blue the first suspicion of plague among the ground squirrels was aroused in 1903, when an epizootic affected so many of the rodents in Contra Costa County, California, that they were almost exterminated. At about the same time, probably later (the history is not definite), there was a heavy mortality among the ground squirrels in Alameda County. . . . There is no proof that the epizootic was plague, but as several cases of plague in man occurred in the territory invaded at about the same time, it seems not unlikely that this disease was present among the rodents. The victims were persons who had not been exposed to the possibility of being infected from a known epizootic among rats. This, coupled with the fact that several of these persons were known to have handled squirrels a few days before the onset of the symptoms, led to the suspicion that plague was prevalent among the rodents."

Actual proof of the above was brought forward in summer 1908, when a squirrel found dead near the house of a plague victim was examined by Wherry<sup>(23)</sup> and independently by McCoy<sup>(24)</sup> with positive findings. A detailed description of McCoy's researches will consume too much space but the conclusions summarized in 1921 by himself<sup>(25)</sup> may with advantage be quoted: "Squirrel plague has been studied intensively for over ten years and we know that plague in these rodents presents some rather unusual features as compared with the

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(20) American Jl. Hyg. 1921, No. 2, p. 182.

(21) Local Gov. Board Rep. 1908/09, p. 20.

(22) Jl. of Hyg. Dec. 1910, pp. 590-592.

(23) Jl. of Infect. Dis. 1908, No. 5, pp. 485-506.

(24) U. S. Publ. Health Rep. 1908, No. 37, pp. 1239-1323.

(25) Amer. Jl. of Hyg. 1921, No. 2, p. 187.



disease in rats. In a large number of cases the disease tends to assume a subacute or chronic form, and doubtless many infected squirrels recover. The lesions induced in guinea-pigs by artificial infection from squirrels also tend to be subacute."

The parasites of the ground squirrel are herewith given. "*Fleas*: Ground squirrels are usually found to be heavily infected with fleas, the commonest species being *Ceratophyllus acutus* Baker; less frequently, the *Hoplopsyllus anomalus* Baker is found. We have shown that the former of these is capable of carrying plague among ground squirrels. No experiments have been performed with the latter. Both of these fleas readily attack man under experimental conditions and indeed also under natural conditions. At one time our squirrel stock room became so heavily infested that upon going into the room one was certain to be bitten by many of the parasites. It finally became necessary to use a pulicide upon the floor in order to make it possible to enter the room without having to suffer the attack of the insects. *Lice*, *mites* and *ticks* are also found, the latter apparently only in certain seasons, and possibly only in limited areas. Our observations on this point are not completed. Of internal parasites a *cysticercus* embedded in the muscles is occasionally encountered. Round worms and flat worms are not rare in the intestine." (McCoy<sup>26</sup>). About 17 human cases have been traced to the squirrels. It is noteworthy that the primary buboes are almost always in the axilla, "due, of course, to the fact that in the handling of infected squirrels, dead or alive, the fleas attack the 'upper extremities', while, when the disease is contracted from rats, the chances are that infection will be through the skin of the feet or legs, resulting in primary buboes in the femoral region." (27).

This corroborates our Harbin findings in human cases directly resulting from tarabagan infection, where axillary buboes also predominate. Two more important points may be emphasised:

- (I) A pneumonic outbreak among 13 persons arising from a human bubonic case of squirrel origin which developed secondary pneumonia that spread through four passages in man, in California in the autumn of 1919<sup>(28)</sup>.

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(26) Jl. of Hyg., l. c., p. 593.

(27) Amer. Jl. of Hyg., l. c., p. 187.

(28) Ibidem, p. 191.

- (II) There is some evidence that human infections due to squirrels are less fatal than those of rat origin, and the clinical picture may be that of a subacute process more often than what occurs in cases infected from rats.<sup>(29)</sup>

Such a case of subacute plague dying in 16 days "with necrotic foci in the kidneys and liver, a large number of caseous nodules up to a walnut in size in the lungs, and multiple caseo-purulent lymph glands" was described by McCoy and Wherry<sup>(30)</sup>.

The American authorities fearing the re-infection of urban rats from diseased squirrels introduced measures for their wholesale destruction. The methods adopted were:—

- (1) poisoning with strychnine, etc.,
- (2) poisoning with carbon bisulphide by means of a special pump.

7. *Wood-rat*.

8. *Field rat*. Only one animal of each species has been found infected under natural conditions. "A field rodent Louisiana," referred to by McCoy<sup>(31)</sup> is probably *Hesperomys pulustris* (New Orleans).

9-11. (*South Africa*). Plague is apparently of recent date in South Africa having been introduced<sup>(32)</sup> during the Anglo-Boer War, 1900-1902. Among the wild rodents<sup>(9,10)</sup> the pest seems to have existed since 1916<sup>(33)</sup>, but it was not until 1921 that the existence of natural plague among them was scientifically established. Plague in the "striped mouse"<sup>(11)</sup> had been found during the early outbreaks in the vicinity of some towns, especially Knysna<sup>(34)</sup>.

Definite proof was difficult to obtain because the carcasses were quickly devoured by small carnivora, birds and ants, and also because of the nocturnal habits of the gerbille. The multimammate mouse occupies in South Africa the position of the ordinary domestic mouse in other countries and lives partly in burrows of the gerbille, partly in dwellings and out-buildings or at least near them. The first infected animals were found at a spot in the open fields where a farmer used

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(29) McCoy, Amer. Jl. of Hyg., 1921, p. 187.

(30) Jl. Inf. Dis. 1909, pp. 670-675.

(31) McCoy, Amer. Jl. Hyg., 1921, p. 187.

(32) Jl. of Hyg., Dec. 1921, p. 377.

(33) Ibidem, p. 380.

(34) „ p. 377.



to have his midday siesta. This individual caught plague and subsequent investigations of this focus revealed an epizootic. The other rodents in this country have so far not been found infected, although one variety, the large-eared mouse (*Malacothrix*) was at one time equally suspected.<sup>(35)</sup> According to Haydon<sup>(36)</sup> rodents other than the gerbille and multimammate mouse were examined but these were too few to permit any definite conclusion. Their susceptibility to artificial infection has not been investigated.

Mitchell<sup>(37)</sup> states: "the original source of infection of the wild rodents in the north-western Free State must remain a matter of surmise. Natives from Tarka, Queentown and neighbouring districts of the Cape Province sometimes go to work on the farms in the Free State, and the infection may have been introduced in 1916 in this way and conveyed from man to rodent by fleas. On the other hand, however, it seems well within the bounds of possibility that the infection has existed amongst the wild rodents in certain parts of the Union since the outbreaks of 1903. . . ."

We have thus in California and North America two interesting examples of the formation of a plague focus, which for all practical purposes may be considered as a "primary" one, evidently of recent date and somewhat definite origin. The possibility of plague arising in a country infested by large numbers of free living (wild) rodents through the rat population should therefore be borne in mind.

The gerbille was found infected in Tunis,<sup>(38)</sup> while the *multimammate mouse* introduced in bags of rice in 1919 led to an outbreak in the Bukedi District (Uganda)<sup>(39)</sup>. 4.3% of those rodents, recently examined in Dakar (Senegal), showed plague<sup>(40)</sup>.

Mitchell gives a complete list not only of the rodents living in South Africa but also of their fleas (see table of fleas). The eradication of these animals was debated but the difficulties were found to be too great. While carbon bisulphide was considered the best poison, Haydon<sup>(36)</sup> pointed out that its fatality to fleas was not so great as to their hosts. The previous destruction of jackals, lynxes and cats which are natural foes of rodents, was found unwise as the latter increased in numbers<sup>(41)</sup>. (Similarly, it was observed that in

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(35) Ibidem, p. 380.

(36) Lancet, Nov. 26, 1921, pp. 1103-04.

(37) J. of Hyg., 1921.

(38) Gobert, Arch. Inst. Pasteur de l'Afr. du Nord, 1921, pp. 440-446.

(39) Uganda Protect. Annual Med. and San. Report for 1921, p. 96-99.

(40) Leger and Baury, Bull. Soc. Pathol. Exotique, Febr. 1923, p. 136.

(41) Mitchell, l. c., p. 379.

the province of Don Cossacks the extermination of foxes (korsaks) proved favourable to the propagation of the susliks<sup>(42)</sup>).

12. *Gold Coast*. Graham gives<sup>(43)</sup> a detailed description of *Cricetomys gambianus*<sup>(12)</sup> and its parasites (see flea table) adding that "it is the rat most frequently met with in native dwellings" and that it appears "at least probable that plague began among *Mus decumanus* and subsequently extended to *Cricetomys gambianus*, a fortnight being the interval separating the respective epizootics of the two species."

11-14. (*East Africa*). *Pelomys fallax tredescens* and *Mus (Thamnomys) aff. dolichurus* were found plague-infected during an epidemic near the Kilimandsharo mountains where pneumonic cases preponderated<sup>(44)</sup>. Among 2893 *Pelomys*, which in this region were domestic, 0.8% were found infected. Among 3294 tree rats (*Thamnomys*) only 0.03% showed plague.

15-17. (*India*) Indian squirrels (*m*) were found by Simond<sup>(45)</sup> in 1898 to be infected at Karachi. This was afterwards fully confirmed by Miss Corthorn<sup>(46)</sup>. Haffkine<sup>(47)</sup> also emphasized their importance. In one instance an English child in India received infection from the bite of a plague infected squirrel<sup>(48)</sup>.

Recently plague infection has been found among the squirrels in Ceylon. Among other findings "squirrel plague has been recorded from Ginigathhena, in 1917 in association with the Nawalapitiya outbreak." No fleas have so far been found upon them<sup>(49)</sup>. They played in this outbreak only a secondary role, rats being its cause, as the Colombo Public Health Department kindly informed us.

A palm rat found infected at Senegal (Africa)<sup>(50)</sup> belongs probably to this species.

It has been ascertained in Belgaum and other places in the Bombay presidency, that a large rat, the *bandicoot*<sup>(16)</sup>, once a common frequenter of town houses, disappeared completely with the advent of plague. No reliable evidence of migration could be obtained, and it is highly probable that this animal—susceptible to plague under laboratory conditions

(42) Loc. Gov. Board Rep. 1912/13, p. 73.

(43) Graham in Simpson, Rep. on Pl., Gold Coast, 1908, pp. 21-25

(44) Lurz. Arch. f. Schiffs-u. Tropenhyg. 1913, No. 17, pp. 593-599.

(45) Annales de l'Inst. Pasteur, 1898, p. 664.

(46) Indian Med. Gaz., March 1899.

(47) Lancet, Feb. 4, 1899.

(48) Farrar, B. M. J. 1902, Aug. 16.

(49) Colombo Rep. 1922, p. 41.

(50) Laveau, Bull. Soc. Path. Exot. 1919, pp. 291-96.



—became almost extinct through natural infection<sup>(51)</sup>. A similar variety of the bandicoots<sup>(17)</sup> found in Calcutta is believed by some observers<sup>(52)</sup> to be intimately concerned in the spread of the disease in this city.

18. (*Ceylon*). The bandicoot of Ceylon has also been known to be affected with plague on a few occasions<sup>(53)</sup>.

19. *India*. In an epidemic at Mysore (1899) several porcupines kept in the zoological gardens died of plague together with some monkeys. This is the first and only record of infection among these animals<sup>(54)</sup>.

20. (*Guinea-pig*). The ordinary guinea-pig (*Cavia cobaya*) has been so extensively used for experimental work that it is not surprising to hear of accidental outbreaks of plague occurring among them. Both the Indian Plague Commission<sup>(55)</sup> in 1906 and Schoebl<sup>(56)</sup> in 1913 recorded such instances, where rat fleas were discovered to be the transmitters. Another outbreak was recorded by Noc<sup>(57)</sup> in 1919.

On three occasions (one in Sidney 1902<sup>(58)</sup> and twice in India 1903 and 1905)<sup>(59)</sup> guinea-pigs were attacked in the zoological gardens. In the first case other animals also succumbed, while in the two Indian instances the infection was traced to rat fleas.

21-22. (*England*). Beside accidental laboratory infections along with guinea-pigs occurring in India in 1906<sup>(55)</sup>, rabbits have been known to suffer from true natural plague. An old report of the year 1680<sup>(60)</sup> mentioned the rabbit as a possible agent in the spread of the pest. Martin and Rowland<sup>(61)</sup> examined 40 rabbits in East Suffolk in winter 1910 and found natural plague in two. In the previous September four human cases of pneumonic plague had been diagnosed in the same county. "It is more than probable that the introduction of plague into East Suffolk was due to grain ships from infected ports arriving in the River Orwell

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(51) Indian Pl. Comm. Jl. Hyg., Pl. No. 1907, p. 760 and Jl. Hyg., Pl. No. 1910, p. 459-460.

(52) Hossak, Jl. and Proc. Asiat. Soc. Bengal, New Series, Vol. V. (1905).

(53) Philip and Hirst, Jl. of Hyg. 1915, p. 543.

(54) Loc. Gov. B. Rep. 1898/01, p. 317.

(55) Jl. Hyg. Vol. VII, 1908, p. 891.

(56) Phil. J. of Sc. 1913, p. 417-421.

(57) Rep. sur le fonct. du Lab. de Bact. de l'Afr. O. F. en 1919, Dakar 1920.

(58) Thompson, Rep. on Second Plague Outbreak in Sidney 1902.

(59) Liston, Jl. Bombay Nat. Hist. Soc. 1905, Vol. XVI, p. 253.

(60) Kijanitzin, Russki Vrach 1910, pp. 1510-12.

(61) Observations on Rat Plague in East Suffolk, 1910.

for Ipswich, and from which vessels rats came ashore and infected the local rodents with plague" (Bruce Low)<sup>(62)</sup>. Browne reported a case of bubonic plague (1911) in a seaman at Shotley, East Suffolk. He had evidently been infected by one or other of the two rabbits handled by him. It was noticed that rat plague was prevalent in this district at the time.

Again in 1914 a dying rabbit picked up in the same locality was bacteriologically proved to suffer from plague. A rat epizootic was also then in evidence<sup>(63)</sup>.

A limited outbreak on board the British S.S. "Bellailsa" occurred in 1912<sup>(64)</sup>, where three apprentices succumbed. It was surmised that the victims received infection from the pet rabbit kept by them. There was an epizootic among the rats on the steamer.

KURAOKA<sup>(65)</sup> recorded an instance at Kagi (Formosa) where many rabbits died during the plague period, but unfortunately no bacteriological examination was made.

22. *Hare*. The only two records available of natural plague among wild hares came from England, both occurring in 1910 in East Suffolk<sup>(66,67)</sup>.

23-24. (*Senegal*)<sup>(23)</sup>. The African bush-rat (*Golunda campane*) was recently found by Leger and Baury<sup>(68)</sup> infected with natural plague in 52 out of 715 examined (7.2%).

24. Laveau mentioned infection in a large field rat in Senegal (1919).

25. (*Tunis*). Besides gerbilles, plague stricken field rats were found in Tunis in 1920<sup>(70)</sup>.

26. (*Rhodesia*) *Field rat Rhodesia*: Kinghorn<sup>(71)</sup> related an epizootic among *Mus rattus* in Rhodesia (1918), in which *Mus norvegicus* and probably field rats were also involved.

(62) Loc. Gov. B. Rep. 1914/17, p. 146.

(63) Statistical Rep. on Health of Navy, 1912, Oct. pp. 173-176.

(64) Harker, Annual Rep. of Med. Off. of Health, Tyne Port San. Author. 1913, pp. 24-26.

(65) Rep. of Saigon Conf. Trop. Med. 1913, p. 207.

(66) Martin and Rowland, Observations on Rat Plague in East Suffolk 1910.

(67) Bulstrode, Loc. Gov. B. Rep. 1910/11, p. 36.

(68) Bull. Soc. Pathol. Exotique, Feb. 1923, p. 136.

(69) Laveau, Bull. Soc. Path. Exot. 1919, pp. 482-484.

(70) Gobert, Arch. Inst. Pasteur de l'Afr. du Nord, 1921, pp. 440-446.

(71) Kinghorn, quoted Trop. Dis. Bull. XIII, p. 324.



27. (*Persia*). Grekoff isolated *Bac. Pestis* from field rats near Turbat-i-jam (*Persia*) in 1912<sup>(72)</sup>.

N.B. The reference about *arvicanthis niloticus* (Egyptian rat) is rather contradictory. While on page 20 of the Report it is stated to be only experimentally susceptible, later on in the same Report (p. 23) the following sentence appears: "In Komombo however, *Mus norvegicus* and *Arvicanthis niloticus* are the rodents which convey the infection to man."

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### III. RODENTS SUSCEPTIBLE TO EXPERIMENTAL INFECTION.

All necessary information upon this subject is outlined in the accompanying Table II.

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### IV. NOTE ON THE PLAGUE PROBLEM IN TRANSBAIKALIA.

Transbaikalia being the endemic centre from which the principal epidemics of pneumonic plague have arisen, a survey of the plague problem in this part of the world is called for.

A detailed description of the human outbreaks which have occurred from time to time in Siberia would be beyond the scope of this paper. The problem, as it affects the rodents, and particularly the tarabagan, may be herewith discussed.

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### V. RODENTS OF TRANSBAIKALIA.

A paper on "Rodents in Mongolia and Manchuria" was read by Woerster at the Mukden Conference (1911), but this was unfortunately not published in the Proceedings<sup>(74)</sup>.

Jetmar<sup>(75)</sup> recently tabulated the rodents of the Dauria Steppes as follows:—

(See Table III, Rodents of Transbaikalia.)

#### *Remarks upon Table III:*

1. Only *Mus musculus* and *Mus norvegicus* have been found among the domestic species. Opinions differ as to their frequency, but no one has yet described any plague infection among them.

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(72) Clemov, Lancet 1913, June 14, p. 1697.

(73) Petrie, Progress Rep. on the work of the Plague Invest. Staff in Upper Egypt, Cairo 1912, p. 20.

(74) Rep. Mukden Conference. p. 336.

(75) Jl. Transbaik. Med. Soc. 1922, No. 2, pp. 95-105.

2. The hamster (*Cricetulus furunculus*) and rat hare (*Ochotoma*) have been found susceptible to experimental infection (see Table II). As the reports have not appeared in any English publication<sup>(76,77)</sup>, they may with advantage be quoted here:

- a. Two hamsters were inoculated percutaneously with B. P. but survived. Out of four inoculated subcutaneously, two succumbed on the 5th day to plague showing besides inguinal buboes, peritonitis and enteritis. The other two survived.
- b. Two rat hares were inoculated subcutaneously with B. P. One died next day accidentally of pneumococcal infection, the other on the 4th day of plague showing local reaction and enlarged spleen.
- c. Three hamsters and two rat hares received injections of B. P. All died within 12-48 hours of plague.
- d. Zarianoff (Manchouli 1923) inoculated intraperitoneally a rat hare with a highly virulent culture of B. P. Animal died of plague after 60 hours.
- e. Generally speaking, percentage of infection for *Ochotoma* 50% and for hamster 25%<sup>(78)</sup>.

The above may throw some light upon the possibility of their playing a role in the epizootology of plague, especially as both species abound in the tarabagan regions and have their burrows in the immediate vicinity of the latter. In our many expeditions we also noticed this feature.

One human outbreak in Matsievskaja (1919) could with some probability be traced back to a hamster. Thus: A married Russian couple lived in a slaughter compound. Their pet cat brought in a dead hamster from the fields and left it on their bed. The woman returning some hours afterwards removed the corpse. Within a week, the husband fell sick with buboes in both groins, while the wife showed swellings in right axilla and left groin next day. Both victims died, showing at post mortem all signs of plague. This incident may help to elucidate the part taken by the cat as a possible transmitter of infection—an aspect not generally realised.

3. As seen in Table I, the suslik (*Spermophilus*) is highly susceptible to B.P. and has given rise to several human outbreaks in the Kirghiz Steppes. It is strange that the varieties encountered in Transbaikalia and Manchuria have up to now been found free from the disease, though one variety (*Spermophilus Eversmani*) is highly susceptible<sup>(78)</sup>.

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(76) Ibid.

(77) Sukneff, Publ. Harbin Med. School, No. 1, 1922, pp. 213-34.

(78) Jetmar. ! c. and Zeit. fur Hyg. and Infekt, 1923, p. 329.



An experiment performed by Sukneff and Jetmar with lice from *Spermophilus dauricus* may be quoted<sup>(78)</sup>. Thirty lice were allowed to feed on the head of a plague-infected guinea-pig. On the death of the latter next morning, two lice were recovered. In one of these, numerous plague bacilli (40,000) were seen. The particular species of louse has not been definitely identified, but it is closely allied to that found on the tarabagan.

4. The jumping hare is a nocturnal animal and is difficult to observe and catch alive. We have seen dead specimens on the fields during our research expedition in Siberia.

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## VI. THE TARABAGAN.

### 1. *Historical survey of natural plague.*

It is unfortunate that until recent years observations upon the tarabagan have been chiefly made by native hunters and not by trained medical men.

Marco Polo thus described this animal in his "Travels": "Tartars subsist entirely upon flesh and milk, and a certain small animal, not unlike a rabbit, called by our people Pharaoh's mice, which during the summer season are found in great abundance in the plains."

Both Bell (1691-1780) who travelled in these parts in 1718-22 and Du Halde, also about the same time, left good descriptions of the animal. The former said<sup>(79)</sup>: "On these hills (near the Selenga river) are a great number of animals called marmots, of a brownish colour, having feet like a badger and nearly of the same size. They make deep burrows on the declivities of the hills; and it is said that in winter they continue in these holes, for a certain time, even without food. At this season, however, they sit or lie near the burrows, keeping a strict watch, and at the approach of danger rear themselves on their hind feet, giving a loud whistle and then drop into their holes in a moment."

Du Halde<sup>(80)</sup> gives a similar description and mentions the catching of the animals in great numbers—"on en prend à la fois un très-grand nombre."

It is hardly necessary to state that the reports emanating from country peasants as to the existence or not of plague should be received with caution. One sign considered by

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(79) Bell's "Travels", publ. in Glasgow 1763, Vol. 1, p. 311.

(80) History of China, Vol. IV, p. 30.

them as characteristic of pest is the presence of a red swelling in the axilla. Even Russian doctors are sometimes misled by this statement, taking the swelling for buboes and class cases so reputed as plague.

In our investigations we have gone into this matter carefully and found the so-called swellings to be merely deposits of fat, often of a pink colour. But this is no more characteristic of plague than of other diseases and is frequently present in healthy animals. A few remarks may now be made of scientific observations made in the past upon this subject. We begin with a classification of the tarabagans believed by us to have actually suffered from natural plague.

It was a Russian military physician who first described what he thought to be an epizootic among the tarabagans in 1901. His words were<sup>(81)</sup> "As I was returning from the village Suktui into Aksha, I saw plague affected tarabagans. The animals were moving unsteadily, their eyes were dim, and they were apparently dazed, inasmuch as, while crawling across the road, they came sometimes under the wheels of the carriage." Unfortunately no bacteriological advantage was taken of this unique opportunity.

It should be pointed out, however, that although an unsteady gait on the part of these animals while in the wild state suggests the possibility of plague, we have often seen similar symptoms present in tarabagans dying of other diseases. On the other hand, quite frequently plague infected animals remained strong and fierce up to the last moment of their demise.

The first authentic case of natural infection was that of Barikin, <sup>(82)</sup> who shot in the autumn of 1907 what he considered a healthy tarabagan in the district between Lake Dalai Nor and Manchouli. His attention had been drawn to this neighborhood by the Mongols who regarded the hills around as infected and therefore avoided them. Not far from this locality plague had occurred in a girl (aet. 13) who probably received infection from a dead tarabagan. The animal when alive looked sick and was killed by her brothers in the field. The girl was ordered to remove the skinned corpse, but it being somewhat heavy, she dragged it along the grass and returned the same way to the camp. She had a small open wound in her left foot, through which the bacilli undoubtedly entered, for she developed a left inguinal bubo afterwards and died of plague. The brothers who cut up the animal escaped.

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(81) Kokosov, quoted by Podbielski Russian Archive for Path., etc., 1901, No. 3, p. 250.

(82) Russki Vrach. 1909. No. 15, pp. 538-540.



The tarabagan shot by Barikin, as mentioned above, was carefully dissected by him two hours after death. The following table indicates his findings:

TABLE IV.

Organ.	Measurements	Cf. normal measurements.	Lesions found.
Heart	8.5 cm. (circumf.) 4.5 cm. (height)	8.0 cm. 4.0 cm.	Muscles ruptured and haemorrhagic.
Liver	16×12×2 cm.	14×9×1.5 cm.	swollen and congested.
Spleen	14×3×1.5 cm.	10×2×1	swollen and congested showing small greyish nodes on section.

(No buboes noted)

Smears from liver, kidney, heart and lung were sterile.

Smears from spleen showed among others suspicious bipolar gram-negative bacilli and involution forms.

Cultures from spleen were suspicious for plague and showed non-motile organisms like B. P. No animal experiments were done.

Histological sections of spleen showed plague bacilli.

Barikin also laid stress upon the similarity of these nodules in the spleen to those found in other rodents. Although some observers have questioned the accuracy of his diagnosis, we have no doubt that this was a true case of plague, partly because of the strong *prima-facie* evidence and partly because we have ourselves observed similar nodules in artificially infected tarabagans.

The *second and third* authentic cases were found in 1911.

As stated in our 1911-13 Reports, <sup>(83)</sup> five suspicious cases were encountered in 1911, of which two proved positive, one doubtful. These were:—

- a. Zabolotny's <sup>(84)</sup> picked up alive by an assistant three versts from Sharasun, but dying soon after. I personally saw the organs in 1911. There were marked signs suggesting plague in the spleen and lungs besides cervical buboes. Pure cultures of B. P. were obtained from all organs. A healthy tarabagan inoculated through the right leg developed two large buboes in the right groin and died soon after.

(83) N. M. Serv. Rep. 1911-13, p. 20.

(84) Archiv. f. Schiffs- u. Tropenhyg, 1912, Beiheft I.

- b. Bissemsky's <sup>(85)</sup> one was found sick in June near Arabulak and died 20 minutes after capture. This animal was semi-conscious, not attempting to escape but coming towards him instead of running away. The hind legs were paralysed (as in our experimental observations). At p.m. haemorrhage in peritoneal cavity were found, the spleen enlarged three times, liver haemorrhagic but no buboes were present. Smears and cultures were positive as well as experiments upon mice.
- c. Bissemiski reported another animal (June 17th) with rt. axillary bubo and positive findings in smears and cultures, though gp. and mouse experiments proved negative.

The *next four* naturally infected tarabagans were found in 1921 by Sukneff. <sup>(86)</sup>

In September two dead animals were picked up in the valley Kinkija (5 versts south-west of Sektui.) Both showed ample evidence of plague, guinea-pig experiments being positive. Sukneff stated that on one of these tarabagans a flea was found, and on the other three lice, in all of which plague-like bacilli were seen.

At the same time these animals were examined, the adjoining field showed numbers of corpses, which had more or less been devoured by beasts of prey, e.g., eagles, polecats, wolves (?). Dr. Krotkoff of the party actually chased an eagle (*tarabashine*—local variety, feeding partly upon tarabagans) found eating a dead tarabagan. Only some flesh attached to the vertebral column was left, but smears showed bipolar-stained bacilli. This case is highly suspicious. On September 22 the fourth tarabagan was found in the valley Barun-Zasulan. Suspicious B. P. were diagnosed in large numbers in the cervical gland. Here also signs suggesting the presence of an epizootic were noticed, e.g., bones, decomposed skins, lying on the ground.

## 2. LATEST FINDINGS OF NATURAL PLAGUE.

Towards the end of May 1923, information reached us of two fatal cases of bubonic plague in man, one near Yakoshih (a village lying on the Chinese Eastern Railway between

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(85) Jasienski and Chmara-Barshevski, Russian Plague Rep. Harbin 1912. Supplement.

(86) Sukneff, l. c.

N.B.—This publication reached us too late to refer to in our Report 1918-22.



Mentuhö and Hailar) and the other at Haranor (in Transbaikalia). These two localities are separated from each other by a distance of 330 versts (210 miles) Haranor being 79 versts west of Manchouli and Yakoshih 25½ versts east of Manchouli. Both victims were Russians, who had hunted tarabagans and developed buboes in the axilla. We also received news of an epizootic among the rodents at Söktui, and hastily equipped an expedition led by three physicians (Wu Lien Teh, Pollitzer and Kwan) to the affected region, hoping to witness personally the existence of plague among the wild tarabagans. Should our mission succeed, we would go a long way in finally establishing the relationship between the Manchurian outbreaks and this interesting animal. In this respect our hopes have been realised as the following pages will show.

The Chinese party started from Harbin on June 2nd, conducted investigations for a week at Manchouli and arrived at Söktui (Siberia) on June 10th. We returned to Harbin on June 23rd, having spent the interval in the suspected areas.

Throughout our tour in Siberia we worked in close co-operation with the Russian Plague Detachment under Sukneft, in whom we found a keen, friendly and experienced investigator. This doctor reported some findings before our arrival. The results of our joint work may be thus summarised:—

- T. 9. One suspicious tarabagan was found on April 29 in the valley Chistokina, 1½ versts south-east of Söktui. P.M. made on May 1st showed no buboes, slightly swollen soft spleen, congested areas in lungs and hæmorrhages in wall of stomach. Films from lungs showed gram-negative, bipolar bacilli. Other organs negative. Cultures and experiments negative.
- T. 10. On June 4th four corpses were found in the Barun Zasulan (see 1921.) This valley is situated ten versts s.e. of Söktui, but on higher elevation. Three were too decomposed for examination, but the fourth proved positive. P.M. showed left cervical bubo. Smears and cultures confirmed plague. Two guinea-pigs inoculated with juice from the organs succumbed to typical pest. Our party helped in the autopsies.
- T. 11. On June 8th a corpse was found in the same valley. No buboes were present, but spleen was soft and swollen with small greyish nodules. Hæmorrhagic foci in lungs (especially left) and swollen suprarenal glands. Films and cultures confirmed plague, while animal experiments in which our party took part also gave positive results.

T. 12. On June 10th our party independently dissected a complete tarabagan found the previous day in the same valley. At P. M. anterior cervical region was slightly swollen. As the skin was drawn aside, a large superficial bubo was seen on left side of neck. Salivary glands were oedematous and congested. The deep cervical glands on left side were also much enlarged up to size of Windsor bean and showed on section caseo-purulent matter. Glands on right side were swollen but contained no pus. Spleen was enlarged and soft. Liver was swollen and congested. Some clear liquid in pericardial sac. Both lungs showed congested areas, especially on right where practically the whole lower lobe was affected. Smears were positive in all organs.

Cultures and guinea-pig experiments were fully positive.

#### HISTOLOGICAL FINDINGS OF T. 4.

A short summary is herewith given:—

- a. *Cervical bubo.* A portion of the caseating area cut. Marked leucocytic infiltration adjoining the caseating region. Isolated masses of B. P. seen in and around Malpigh. bodies. Haemorrhages in lymph spaces. Capsule of gland infiltrated.
- b. *Salivary gland (submax).* Intense infiltration. Haemorrhages into and around acini of glands. B. P. in small numbers among the lobules. Majority of epithelial cells of acini show granular degeneration. Lymph spaces invaded by red cells.
- c. *Lungs.* Portion of congested area of right lung cut. Marked early pneumonia seen. Most alveoli invaded by red cells and leucocytes, others collapsed. Numerous B. P. in alveoli and interalveolar spaces. Proliferation of cells of infundibula. Intense reaction.
- d. *Spleen.* Lymph nodules darkly stained. B. P. in great numbers in and between Malpigh. bodies. Capsule infiltrated. Blood vessels dilated. Red cells invaded lymph spaces.

T. 13. On June 23rd, (i.e. after our departure) a decomposed tarabagan was found in the same valley. Guinea-pig inoculated with material from this died of typical plague.



T. 14-16. Three more naturally infected tarabagans were found on July 4, 21 and 25, 1923. Of these, two specimens (14 and 15) were partly devoured, and diagnosis was made from smears of remaining flesh. The third carcase (T. 16) was complete and displayed all typical features, especially cervical buboes and lung lesions. Animal experiments were positive. This specimen is now in our Harbin museum. Photo herewith.

Sukneff and our party discovered two more naturally infected tarabagans (T. 17 and 18), in the autumn of 1923.

The first animal was found in the old infected valley Barun-Zasulan on August 24th, 1923, while the second was found near Dauria where no infected animal had hitherto been recorded (Sept. 1st.)

T. 17. Showed the following p.m. features:

*Glands*:—Cervical glands of both sides much enlarged and congested; no surrounding inflammation.

*Lungs*:—Both sides markedly congested with pneumonic foci all over.

*Other organs*:—Showed acute congestion.

*Smears*:—Were all positive, but lungs showed more B. P. than elsewhere, particularly cervical glands.

T. 18. (Partly eaten by eagles) displayed all features of chronic plague (nodular variety, like No. 1.)

Left *submaxillary bubo* present—size that of a cherry with little hyperemia and no surrounding inflammation.

*Cervical glands*—only slight changes.

*Lungs*—pinkish colour, some bulging nodes with no signs of acute inflammation.

*Liver*:—Enlarged three times normal, extremely congested, with many yellowish-white bulging nodules and haemorrhagic spots.

*Gall bladder*—enlarged.

*Spleen*—mostly eaten off. No evident swelling.

*Smears*—B. P. present—but scantily—in all organs.

*Animal experiments*:—Gp. inoculated with fresh blood from lung and heart succumbed in 5 days to acute plague. Culture from liver showed considerable diminution of virulence within short period.

Cultures of *B. P.* from both tarabagans satisfied all confirmatory tests.

One isolated fatal case of bubonic plague (rt. axillary bubo) occurring in a girl, aet. 12, on September 3rd, near Dauria is noteworthy. This case could be traced back to a hunting camp not many versts distant from the place where we found T. 18.

All the evidence hitherto obtained seems to point to the fact that epizootics, when they occur, are usually confined to a limited area. The rise and decline of such outbreaks have still to be studied. Although satisfactory evidence had been collected regarding the 1923 cases of natural plague tarabagans, it was nevertheless necessary in addition to test the cultures by all methods so as to exclude other organisms similar to *B. Pestis*, particularly *B. pseudotuberculosis rodentium*.

Four strains were brought back by us from the Transbaikalia cases to our Harbin Laboratory. These were:—

- |    |   |       |
|----|---|-------|
| a. | Culture bubo from gp. infected cutaneously with material from .....   | T. 10 |
| b. | Culture spleen from gp. infected cutaneously with material from ..... | T. 11 |
| c. | Culture spleen from gp. infected cutaneously with material from ..... | T. 12 |
| d. | Culture lung from .....   | T. 12 |

From each culture one loop was rubbed into the shaved and scarified skin of a guinea-pig. Every animal died showing typical signs of acute plague.

From these infected guinea-pigs, cultures e,f,g,h, were obtained and together with a,b,c,d, were subjected to the necessary bacteriological tests. The results obtained in every strain were:—

- i. Gram-negative, bipolar stained, non-motile bacilli.
- ii. Typical growths of *B. P.* on ordinary agar and bouillon.
- iii. Formation of involution forms on 3% Salt Agar.
- iv. No change in litmus-milk, except very slight acidity.

The final observations prove conclusively that the organisms in question are true plague bacilli. Consequently, the dead tarabagans No. 10, 11, 12 picked up in the fields of Transbaikalia in June 1923 did really die of natural plague. Similar results were obtained also with cultures from T. 17 and 18.



## 3. WINTER EXPERIMENTS.

Zabolotny and other Russian observers believe that the district of Weichang (lat. 42, long. 118), the famous Imperial hunting park of N. China, is a true endemic centre of plague, basing their conclusion upon the following:—

- i. Mongolian and Buriat pilgrims, on their return from the sacred Chinese mountain of Wu T'ai Shan, passed through this locality and became infected.
- ii. As the disease developed and the victims died during the journey northwards, their bodies were left according to religious custom on the steppes and were thus eaten by tarabagans besides other animals.
- iii. In this fashion perhaps the infection was maintained among these rodents.

Dudchenko<sup>(87)</sup>, who had devoted much time to the study of the tarabagan problem in Siberia, considered that the hibernating habits of the animal helped to limit the spread of the disease, inasmuch as the sick ones usually stayed outside their burrows to die. In trying to assign a reason for the almost yearly appearance of the epizootic among tarabagans, he laid stress upon the regular introduction of plague by the pilgrims passing through Weichang.

It is true that Catholic missionaries reported cases of bubonic plague at Weichang as early as 1888, and Zabolotny on his visit there in 1898 bacteriologically confirmed the disease<sup>(88)</sup>. But since that time nothing has been heard, and Chinese medical officers stationed in the neighborhood have not reported any case for nearly 25 years.

This fact together with our increased knowledge of the epidemiology of plague in humans and rodents, do not support the Russian view, and in our opinion, some other cause must be sought for the propagation of plague among tarabagans. Hence a series of investigations upon the type of disease encountered in winter seems of great importance.

Le Dantec<sup>(89)</sup> was the first to suggest the possibility of *B. Pestis* remaining quiescent in the body of the tarabagan during winter and killing it in spring. Such spring outbreaks have often been reported by Russian and Mongol peasants, but they should not be accepted without sufficient proof, as the animals may become starved in the early cold spring or be attacked by enteritis. It is interesting to men-

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(87) Vestnik Obst. Guig. 1909, pp. 897-909 and 1045-1089.

(88) Russian Archive f. Path. etc., 1899, pp. 242-250.

(89) Jl. de Med. Bordeaux, 1911, No. 13, p. 197.

tion that tarabagans are sometimes seen to come out in the open even before the snow has melted. This may be due to the prevailing winter lasting longer than their hibernating period or to some unusual event happening in the burrows.

Wurtz carried out some plague experiments upon Alpine marmots but they were not successful as the animals awoke from hibernation and died after a few days.

Dujardin-Beaumetz and Mosny<sup>(90)</sup> in 1912 injected B. P. subcutaneously into three European marmots; the two hibernating ones died after 61 and 115 days respectively showing no buboes, but only foci of chronic pneumonia, in which B. P. were present in enormous numbers. The third animal, which was not hibernating, died in  $2\frac{1}{2}$  days. These French observers concluded from this scanty evidence that the tarabagan of Siberia is a "reservoir" for plague, the virus being held in abeyance during winter. They also mentioned that the fleas might serve as carriers and quoted Gauthier and Raybaud who demonstrated the presence of virulent B. P. in the stomachs of fleas after 45 days preserved in the ice box.

Zabolotny and Tshurilina<sup>(91)</sup> also succeeded in 1911 in keeping hibernating tarabagans alive for 12-14 days after inoculation, while controls died in 3-4 days.

Our winter experiments upon over twenty tarabagans in our Harbin Laboratory were continued from November 1922 to April 1923. The external temperatures during these months were as follows:—

TABLE VI.

	<i>Max.</i>	<i>Min.</i>
November 1922 .....	12.5°C.	-29.6
December 1922 .....	-4.1	-27.9
January 1923 .....	-7.4	-33.7
February 1923 .....	-1.6	-30.4
March 1923 .....	13.2	-24.8
April 1923 .....	26.8	- 8.9

In the course of our investigations we had in mind:—

1. The variation (if any) in the temperature of the animals during winter.
2. Continuation of inhalation experiments in winter.
3. Infection by nostrils.
4. Percutaneous inoculation.
5. Inoculation per conjunctiva.
6. Observation upon contacts.

(90) Compt. Rend. Acad. Sci. 1912, Vol. 155, p.p. 329-332.

(91) Berdnikow, Zentralbl. f. Bact. Vol. 69, 1913, p. 257.



It would be tedious to describe fully in this paper our several findings. The methods adopted were along the lines mentioned in our previous publications. Only the salient points will be pointed out here.

- a.* The temperatures differed as greatly in winter as in summer.

TABLE VII.

Temperature	Winter (Recorded 1922-23)	Summer (Recorded 1911).
Highest	37.0 (Normal)	39.4 (Normal)
	39.5 (Plague infected)	40.4 (Abnormal)
Lowest	1.0 (Normal hibernating)	35.0 (Normal)

- b.* In plague infected animals the highest point observed was 39.5, but the fever was rarely continuous, the chart curve sometimes reaching normal and then rising again.
- c.* When the body temperature reached 17.0C, the animals usually became drowsy. Below 14, true hibernation set in.

(See Table VIII—tabulation of winter experiments.)

## 2. CONTINUATION OF INHALATION EXPERIMENT.

Six animals were given inhalations of B. P. from graduated sprays, and then left in unheated rooms.

Of these three were in a hibernating state. The results of spraying obtained when animals were confined in a box or while strapped upon a stage were the same and therefore require no additional remarks. As seen from Table VIII, one (T. 197) did not hibernate but remained feverish all the time until its death 37 days afterwards. At P. M. chronic plague was observed with nodular abscesses in lungs, pericarditis, swollen congested spleen, white patches and haemorrhages over liver, purulent (not bloody) sputum in trachea. The next three animals remained hibernating until their deaths 59, 21 and 41 days respectively after inoculation. T. 223 showed at P. M. haemorrhages and congested areas in lungs. Thymus was swollen and congested, spleen slightly enlarged, peritoneal vessels injected. Smears from all organs showed a few B. P. Cultures remained sterile. T. 233 had bloody froth in nostrils and trachea, cervical glands were swollen and congested. Lungs showed pneumonic areas. Liver had white patches. Spleen was swollen.

Smears and cultures were positive. N.B. This animal did not die until 21 days after inoculation, and yet signs were those of acute plague.

T. 238, dying in 41 days, received same culture as T. 233. Trachea appeared healthy. Lungs were pink with two small congested areas. Spleen was firm, not enlarged, but had some resistant bulging nodules. Liver was congested. Rectal muscles looked haemorrhagic, but were not torn.

Smears from heart doubtful; from spleen nil; from lungs suspicious; B. P. among other organisms.

*N.B.*—The changes in this spleen may indicate resolving plague, as none of our numerous post mortems upon healthy tarabagans have ever shown this feature.

T. 231 and T. 232 were inoculated at the approach of spring when the weather was rather warm. The results were in harmony with those of our previous summer experiments, the lungs showing pneumonia, and the trachea bloody froth.

On the whole, our winter experiments with inhalation indicated considerable prolongation of the disease, as the earliest death occurred 21 and the latest 59 days after infection. This is the more remarkable as the animals were daily handled. We propose to continue similar investigations next winter on a large scale and allow the hibernation to continue uninterruptedly.

### 3. INFECTION BY NOSTRILS.

Two hibernating animals were used. One (T. 224) died in 7 days showing enlarged cervical glands, much bloody froth in the trachea, haemorrhages and pneumonic patches in lung, swollen spleen and congested peritoneum. Bacteriological results were positive.

The second (T. 250) died after 86 days showing absolutely no traces of plague at P. M. There was no doubt about the virulence of the strain employed (Haranor 1922).

### 4. PERCUTANEOUS INOCULATION.

One tarabagan (T. 241) was inoculated on abdominal skin while hibernating. It died after 74 days. At P. M. the right inguinal glands were slightly swollen. Right axillary glands were enlarged, one of which showed a yellow nodule (? caseating area). Deep cervical glands slightly increased in size. Thymus and axillary fat congested. Smears and cultures negative.

*N.B.*—This may be another case of resolving plague. The strain used was virulent Haranor 1922 one.



## 5. INOCULATION PER CONJUNCTIVA.

Two animals (T. 236 and T. 251) received in left eye one loop each from virulent agar culture (1921 strain).

T. 236 developed fever and slight local reaction. It died on 17th day. At P. M., left conjunctiva showed congestion, but there was no gross destruction of eye. Anterior cervical glands partly congested. Lungs congested. Heart surface covered by filmy membrane. Liver, swollen and brown, showed numerous pin-head nodules of yellowish white colour. Spleen soft and swollen with white nodules, size of lentil surrounded by hyperdemic zones. Smears, culture and animal experiments were all positive.

N.B.—This is perhaps a case of the “*nodose*” variety described by Barikin among wild tarabagans. It is also noteworthy that both guinea-pigs infected percutaneously with cultures from this tarabagan survived for nearly two weeks. A white rat, infected subcutaneously, died in 48 hours.

T. 251 died three months after inoculation with app. negative results.

6. *Observation upon contacts.*

Of 4 contacts (three in hibernating stage) used for inhalation experiments, two died. One (T. 213) succumbed after 62 days showing no traces of plague infection. Although most of the time unconscious, it managed in one of its waking moments to eat up part of a plague infected corpse (T. 197). This is of additional interest.

The second tarabagan (T. 234), partly hibernating, also fed upon an infected corpse (T. 232). This occurred 108 days after its first exposure. Five days after the feeding, the animal died showing plague.

At P.M., cervical glands swollen and congested,  
Trachea full of bloody froth,  
Thymus enlarged and congested,  
Lungs pneumonic areas,  
Oesophagus haemorrhages in muscles,  
Stomach and duodenum extensive haemorrhages in  
mucous membrane,  
Spleen soft and swollen,  
Liver few white patches,  
Intestines and mesentery congested,  
Smears and cultures positive.

Apparently T. 234 kept well for 108 days after first exposure to an inhaled mate, but succumbed five days after eating a dead companion, and showed plague with pneumonic complication.

Two other healthy animals were left in untouched box (March 1923) where two plague corpses had just been removed. Both survived.

#### 4. PARASITES OF THE TARABAGAN.

*I: Fleas.* The flea of the tarabagan was first identified by Silantiev, after whom it had been named (*Ceratophyllus Silantievi*).

In 1903 a full description was given by Tiraboschi<sup>(92)</sup> who fully realised its importance in relation to the etiology of plague. Dudchenko<sup>(93)</sup> in 1909 noticed that the fleas under normal conditions did not live upon man, but did bite when suitable occasions arose, resulting usually in red spots sometimes in urticaria on the site of feeding. In a subsequent paper<sup>(94)</sup> Dudchenko (1915) mentioned a case where in the process of digging up a tarabagan burrow one of his workmen was bitten by a flea which turned out to be one from the tarabagan. Two years previously, i.e. 1911 during an expedition in Mongolia, the author W.L.T.<sup>(95)</sup> had already proved in a number of experiments that *Ceratophyllus S.* readily fed upon human beings when hungry. Jetmar<sup>(96)</sup> pointed in 1922 to the presence of apparently free living fleas during the harvesting season in Transbaikalia. He recorded an outbreak of bubonic plague among a group of Russian peasants who had not been in touch with any tarabagan or rodent but had been bitten by fleas when coming in the fields.

As in the case of rats, we have always considered that a knowledge of the flea rate in tarabagans might contribute to the elucidation of the plague problem in these regions. For this reason all data available on this point have been collected and are herewith given:—

1. Petrie<sup>(97)</sup> at the time of the Mukden Conference, April 1911 reported the following figures for fleas transported from Manchouli to Mukden (925 miles).

2,2,2,3,0,2,2,5,2,0,12,3 (12 animals, average 3)

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(92) Archives de Parasit. 1903-04, 8, p. 161.

(93) Vestnik Obst. Guig. 1909, July.

(94) Ibid. 1915.

(95) N. M. Service Rep. 1911-13, pp. 40-43.

(96) l. c.

(97) Report Mukden Conference, p. 237.



2. The author W.L.T.<sup>(98)</sup> while staying in Mongolia, August to Sept. 1911, examined a number of freshly caught tarabagans and found the flea rate to vary greatly. On one animal 94 fleas were caught, but the number seldom went below five. Average was over 10.

3. Occasional observations made by Russians (Wassilewski,<sup>(99)</sup> etc.) also suggested a high flea rate in the autumn.

4. In May-June 1923 we returned to flea statistics and found the following:—

Manchouli, May 19: 20,2,0,1,0,1,0,0, (8 animals, average 3).

Harbin, May 29: No fleas in any of the 14 animals freshly arrived from Manchouli (584 miles).

Manchouli, June 6: 2 tarab. arrived the night before and were kept in a tin bucket. Next morning one was found dead. It had then four living fleas, six ticks and four lice. Strange to say the surviving one had no fleas, only four ticks.

Manchouli, June 4: 0,0,1,0,0 (5 animals, average 0.2).

Manchouli, June 7: 0,3,1 (3 animals, average 1.3), all freshly captured.

Manchouli, June 9: 0,0,0,0,0,1,0,2 (9 animals, average 0.3.)

5. Sukneff (1923) reported as follows:—

Haranor, May 28: 0,0,1 (3 animals, average 0.3) freshly dug from holes.

Haranor, May 30: One shot tarabagan had one flea.

Soktui, June 8: One natural plague-infected tb. showed 3 fleas.

Soktui, June 20: One shot tb. had no fleas.

The above observations support our previous contention that the flea rate in spring is small as compared with that in autumn. This seems important as human plague is usually reported from Siberia towards the end of summer or in autumn. The fact that this period also corresponds to the hunting and harvesting season should not be overlooked. It is also worth remembering that numerous ectoparasites, including fleas, lice, ticks have been constantly found sticking

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(98) N. M. Service Rep., 1911-13, p. 40.

(99) Jl. of Harbin Med. Society, 1921, pp. 27-37.

to tarabagan carcasses and even on skins and pelts which had been removed for several days. This is an important point in discussing the epidemiology of plague in these regions.

6. *Biting experiments.*

In the summer of 1923 we allowed different ectoparasites found on tarabagans to bite human beings, and guinea-pigs.

A summary of this work is herewith appended:—

May 23. Two fleas starved four days were used. One (m) bit man for 31 min. the other refused to bite and died next day.

25. Above flea bit man for 10 min.

26. Same flea bit healthy guinea-pig for 10 min.

29. Tb. louse bit man for 5 min.—Ticks (over 30) were repeatedly tried but refused to bite.

31. Flea of May 31 allowed to bite infected gp. for 5 minutes. Next day it fed on healthy tb. and healthy gp. but both animals survived. This famous flea finally died on June 6th, having been in our possession for 18 days and made 2 journeys between Manchouli and Harbin, that is, travelled 1200 miles. After death the whole body was ground in saline solution and injected intraperitoneally into gp. No plague resulted.

The biting experiments on man were continued at Manchouli in June 1923. The lengths of time intervening before they released their victims were 13,  $4\frac{1}{2}$ , 3, 2. Average 6 m.

In all these experiments we have never seen a flea defecating *while biting*. On only one occasion did a flea deposit its feces on the skin, but it did not bite.

6. *Further experiments done with parasites of tarabagans:*

Three more sets of experiments were performed to transmit plague

a.) from tarabagan to tarabagan

b.) from tarabagan to guinea-pig through bite of fleas or lice. But all gave negative results.

In another set of experiments three lice found on a naturally infected *dead* tarabagan (T. 17) were crushed in normal saline solution and injected subcutaneously into a gp.



This gp. died of acute plague after 6 days. Cultures obtained from the gp. gave further confirmation.

The same kind of experiments carried out with 6 crushed fleas from the same naturally infected tarabagan, gave negative results. Another interesting point is, that the crushed lice showed numerous B.P. under the microscope, but the crushed fleas showed none. (On two other occasions crushed fleas removed from infected tarabagans have given positive results).

The tarabagan flea (*Ceratophyllus Silantieri*) was proved to be a very poor jumper as compared with *Xenopsylla cheopis*.

## 7. CONCLUSIONS.

In view of our researches conducted since 1911, and especially during the last two years, it is practically certain that the series of sporadic outbreaks of plague occurring almost yearly in Transbaikalia, which on two occasions, (1910-11 and 1920-21) invaded Manchuria and caused the death of 60,000 and 9600 respectively, originated in the Tarabagan (*Arctomys bobac*). We have observed that although *Mus norvegicus* and *Mus musculus* are encountered in Siberia and Manchuria, no case of human infection has ever been traced to them. We are now satisfied in the first place that the disease could be produced among tarabagans experimentally in both acute and chronic forms, and in the second place that it exists naturally in both forms.

This being the case, we may next ask ourselves:—

- A. How is the pest conveyed from animal to animal?
- B. How is the pest transmitted to man?

A. Experimental evidence has shown that the tarabagan is easily susceptible to plague by all known methods of infection. The first point that suggests itself to us is the parasitic one. Although we have attempted a number of experiments with their ectoparasites (fleas, lice and ticks), we have up to now failed to obtain positive results. We find the tarabagan an exceedingly strong and vicious animal when compared with the domestic rat, in addition to its wild country habits. Certain important facts may, however, be stated in this connection:—

### (a) *Predominance of cervical buboes.*

Out of eighteen authentic cases of natural infection collected (Table V), we found in eight (nine ?) marked cervical buboes. Three others were only rem-

nants, in which no such records could be made. In the remaining six, no cervical buboes were noted.

(b) *Frequent involvement of lungs.*

Out of our eighteen cases, we find the lungs involved on ten occasions, free on two occasions, and unascertainable in the other six. How far such lesions are primary and how far secondary, we are at present unable to say, but it is interesting to note that our histological examinations often show an acute pneumonic process with much effusion of blood into the alveoli of the lungs.

These observations coincide rather with McCoy's findings among the ground squirrels of California.<sup>(100)</sup> This fact together with the ease with which artificially induced pneumonic plague can spread among tarabagans should be borne in mind.

(c) *Feeding.*

Although the tarabagan is by nature herbivorous, we have often seen the living ones devouring carcasses of their mates kept in the same cage. In fact we have recorded positive transmission by this means. But we shall be on safe ground if we say that this means of transmission, even if it occurs naturally, is very rare.

(d) *Percentage of chronic cases.*

At least three of our eighteen cases record a chronic or subacute condition. This is usually shown in the form of greyish nodules in the spleen, sometimes in the lungs and in the liver, in which B. P. may or may not be seen. Some chronically infected tarabagans in our experiments however, i.e. those living beyond the average period, sometimes died displaying marked acute signs in the organs.

B. When we turn to the question "How is the pest transmitted from animal to man?," we are confronted with four possibilities, namely:—

- a. Eating of the flesh by man,
- b. Inhalation of the organisms by man,
- c. Direct transmission through an open wound in man,
- d. Indirect transmission through the ectoparasites.
- a. Russian observers have for a long time believed that plague in Siberia is caused by the eating of tarabagan flesh. No scientific evidence has been brought to

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(100) American Jl. Hyg. 1921, p. 191.



support this view, although it is sometimes mentioned that the flesh was not thoroughly cooked. The Local Gov. Board<sup>(101)</sup> quoted an incident in which three Russians developed pneumonic plague after eating tarabagan flesh. We have ourselves also received such tales, but on every occasion (particularly when investigating on the spot the so-called pneumonic outbreak of Haranor in 1922) have traced these pneumonic cases to earlier bubonic and septicemic affections.

- b.* It is difficult to see how the hunters can be infected by the living tarabagan when in the fields. The Buriats and Mongols shoot the animal, while the Chinese use the wire trap. Lately the Russians have adopted the Chinese method. Scarcely, if ever, are they in close contact with tarabagans in an enclosed space. After the skinning, the pelt is collected with others in bundles, is stretched out and dried near the camp. No evidence of B. P. surviving on a dried skin has been furnished, and hence the possibility of a hunter being infected by inhalation from the carcass or the dried skin is very remote. There is however, a case on record in 1907 where a Russian dealer in tarabagan skins developed an axillary bubo and died. She had not been on the fields.
- c.* The hunter has never been observed to use gloves, and it is possible that during the skinning operations, when his hands are often chipped, the plague organisms may gain entrance directly into the circulation through the open wound. We have many records of such infections.
- d.* Although indirect transmission through ectoparasites, especially the flea, has not been observed from a naturally infected tarabagan, this is quite conceivable in view of our knowledge of the work upon the rat flea in India.

WU LIEN-TEH.

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(101) Loc. Gov. B. Rep., 1912-13, p. 73.

TABLE I.

LIST OF RODENTS KNOWN TO SUFFER FROM SPONTANEOUS PLAGUE  
OTHER THAN THE DOMESTIC RAT AND MOUSE.

Name.	Locality.	References.
1. Tarabagan <i>Arctomys bobac</i>	Transbaikalia, Mongolia, etc.	Bjeliavski and Rjeshetnikoff, Vestnik Obstchestvennoi Guiguieny 1895.
2. Sisel (suslik) <i>Spermophilus</i>	Kirghiz Steppes	Demiński, Russki Vrach, 1913, No. 30, p. 1069 and Berdnikow, Zentralblatt f. Bakt 1913, vol. 69, pp. 251- 59.
3. Jerboa (family Di- podia, species <i>Dipus</i> )	„	Berdnikow, ibidem.
4. "Wild mouse"	„	Zabolotny, Archiv. f. Schiffs and Tropenhyg. XXVI, 1922, p. 382.
5. "Black Marmot"	Semiretchinsk	Russian Public Health Report, 1907, p. 162.
6. Californian Ground squirrel <i>Citellus beechyi</i> , Richardson	California	Wherry, Jl. of Inf. Dis. 1908, No. 5, pp. 485-506. and Mc- Coy, U. S. Publ. H. Rep. 1908, No. 37, pp. 1289-1323.
7. Dusky footed wood rat (Brush rat) <i>Neotoma fuscipes</i> , Baird.	„	McCoy and Smith, Jl. Inf. Dis. 1910, Vol. VII, pp. 368-73.
8. Field rat <i>Hesperomys pulustris</i>	New Orleans	William, American Jl. Publ. H. Nov. 1920, p. 13 of reprint. (1)
9. Gerbille <i>Tatera lobengulae</i> .	South-Africa.	Mitchell, Jl. Hyg., 1922, No. 4, p. 377-82 and Haydon, Lancet, Nov. 26, 1921 pp. 1103-04. (2)
10. Multimammate mouse <i>Rattus coucha</i>	„	Mitchell, Jl. Hyg., 1922, No. 4, p. 377-82 and Haydon, Lancet, Nov. 26, 1921 pp. 1103-04. (2)
11. "Striped mouse" <i>Arvicanthus pumilio</i>	„	Mitchell, Jl. Roy. Army Med. Corps, 1906, VI. p. 130.

(Continued on next Page.)



TABLE I.—(Continued).

Name.	Locality.	References.
12. <i>Cricetomys gambianus</i> Waterhouse.	Gold Coast.	Graham in Simpson, Report on Pl. in the Gold Coast in 1908, pp. 21-25.
13. <i>Pelomys fallax ire-</i> <i>descens</i> Heller.	East Africa.	Lurz Arch. f. Schiffs and Tropenhyg. 1913, No. 17, pp. 593-99.
14. Tree rat <i>Mus</i> ( <i>Thamnomys</i> ) <i>aff. dolichurus</i> Smuts	"	" "
15. Squirrel <i>Sciurus palmarum</i> .	India. Ceylon.	Simond, Annal. de l'Inst. Pasteur 1898, p. 664. (3) Colombo Report 1922, p. 41.
16. Bandicoot <i>Bandicota indica</i> ( <i>Nesokia bandicota</i> )	"	Jl. Hyg. 1907, Plague No. p. 760 and 1910, Pl. No. pp. 459-460.
17. Small Bandicoot <i>Nesokia bengalensis</i> .	"	Hossack, Jl. and Proc. Asiat. Soc. of Bengal, New Series, Vol. V. (1906).
18. <i>Bandicota malabarica</i>	Ceylon.	Philip and Hirst, Jl. of Hyg. 1915.
19. Porcupine <i>Hydrochoerus capy-</i> <i>bara</i> .	Mysore (India).	Bruce Low, L. G. B. R. 1893/01 p. 317.
20. Guinea-pig <i>Cavia cobaya</i> .	Sidney	Thompson Rep. of the Board of Health on a 2nd outbreak of Plague at Sidney 1902.
	India	Liston, Jl. Bombay Nat. Hist. Soc. 1905, Vol. XVI. p. 253 and Jl. Hyg. 1908, Vol. VII, p. 891.
	Manila	Schoebl. Phil. Jl. of Sc. 1913 pp. 417-421.
	Senegal	Noc. Rep. sur le fonctionnement du Lab. de Bact. de l'A. O. F. en 1919, Dakar 1920.

(Continued on next Page.)

TABLE I.—(Continued).

Name.	Locality.	References.
Rabbit	India	Jl. of Hyg., 1908, Vol. VII p. 891.
21. <i>Lepus cuniculus</i> .	England	Martin and Rowland. Observations on Rat Plague in East Suffolk, 1910.
22. Hare <i>Lepus timidus</i> .	„	Martin and Rowland, Observations on Rat Plague in East Suffolk, 1910 and Bulstrode, L. G. B. R. 1910/11 p. 36.
23. African Bush rat <i>Golunda campaneae</i> .	Senegal	Leger and Baury. Bull. Soc. Pathol. Exot. Feb. 1923, p. 136.
24. "Large field-rat"	„	Laveau, Bull. Soc. Path. Exot. 1919 pp. 482-484. (4)
25. Field-rat.	Tunis	Gobert, Arch. Inst. Pasteur de l'Afr. du Nord, 1912, pp. 440-46. (4)
26. Field-rat.	Rhodesia	Kinghorn, 1918 quoted Trop. Dis. Bull. XIII., p. 324. (4)
27. Field-rat.	Persia	Grekoﬀ, quoted by Clemov, Lanc. '1913, June 14, p. 1697. (4)

- Remarks :
1. Most probably identical with the "field-rodent Louisiana" mentioned by McCoy, l.c. 1921.
  2. Gerbilles were found infected in Tunis as well (see Gobert, l.c.), *R. coucha* in Uganda (see Uganda Protect. Ann. Med. and San. Rep. for 1921, pp. 96-99) and in Dakar (Leger and Baury, Bull. Soc. Pathologie Exot., Jan. 1923. p. 136).
  3. One palm-rat was found infected in Senegal (see Laveau. Bull. Soc. Path. Exot. 1919, pp. 291-96).
  4. The four last-named field-rats were mentioned. because it could not be determined if they as f.i. the fieldrat of Java are a variety of *Mus rattus* (see Swellengrebel. Geneesk. T.v. Ned. Indie 1913, pp. 53-154) or not.
  5. The shrews (*Crocidurae*), often called "Musk-rats," belonging to the order of Insectivora, are not included, although they are known to suffer from plague.



TABLE II.

ADDITIONAL LIST OF RODENTS IN WHICH NO NATURAL PLAGUE HAS BEEN FOUND BUT WHICH ARE SUSCEPTIBLE TO ARTIFICIAL INFECTION.

Name.	Locality.	References.
<i>Spermophilus mongolicus</i> (Citellus) Linn.	South Manchuria	Wu Lien Teh and Eberson, Jl. of Hyg. 1917, pp. 1-11. (a)
Hamster <i>Cricetulus furunculus</i> .	Transbaikalia	Jetmar, Jl. of Transbaik. Med. Soc. 1922, No. 2, pp. 95-108 and Sukneff, Publ. of Harbin Med. School, No. 1, 1922, pp. 213-34. (b)
Rat-hare <i>Ochotoma</i> .	"	" "
<i>Spermophilus Eversmanni</i> Brandt.	"	Jetmar, Zeit. fur Hyg. und Infekt. 1923, p. 329.
Alpine marmot <i>Arctomys marmota</i> .	Europe	Wurtz, quoted by Dujardin-Beaumetz and Mosny; Dujardin-Beaumetz and Mosny, Compt. Rend. Acad. Sci. 1912, V. 155, pp. 329-332. (b)
Field mouse <i>Microtus californicus</i> .	California	McCoy, Jl. Inf. Dis. June 12, 1909, pp. 283-87.
California pocket gopher <i>Thomomys bottae</i> .	"	Ibidem & (c). McCoy, Jl. Inf. Dis. Jan. 1911 pp. 42-46.
Chipmunk <i>Callospermophilus</i> (Citellus) <i>chrysodeirus</i>	"	McCoy, Jl. Inf. Dis. Jan. 1911 pp. 42-46.
<i>Ammospermophilus leucurus</i> , Merriam.	"	McCoy and Chapin, U. S. H. Bull. No. 53, 1912, pp. 15-16.
Rock squirrel <i>Citellus grammurus</i>	New Mexico	McCoy and Smith, Jl. Inf. Dis., 1910. pp. 374-76.
Arizona prairie dog <i>Cynomys ludovician. arizonensis</i> Mearns.	"	" "
Eastern desert wood rat <i>Neotoma albigula. angusticeps</i> Merriam.	"	" "

(Continued on next Page.)

TABLE II.—(Continued).

Name.	Locality.	References.
"Root rat" <i>Tachyoryctes daemon</i> . Thos.	East Africa	Lurz. Arch. f. Schiffs and Tropenhyg. Sept. 1913, pp. 593-99.
<i>Arvicanthis niloticus</i>	Egypt	Todd in Petrie, Progress Rep. on the work of Pl. Invest. Staff in Upper E., 1911-12, Cairo 1912, p. 20. (d)
<i>Acomys cashirinus</i> .	Egypt	Todd in Petrie, Progress Rep. on the work of Pl. Invest. Staff in Upper E., 1911-12, Cairo 1912, p. 20. (d)
Jerboa.	"	" "
<i>Mus agrarius</i> .	Formosa	Kuraoka, Rep. of Saigon Conf. Trop. Med. 1913, p. 204.
Field mouse.	"	" "
Field mouse <i>Arvicola arvalis</i> .		Austrian Plague Report III, pp. 700-701. (e)
Lerot (door mouse). <i>Myoxus murinus</i> .	Senegal	C. R. Acad. Sci. 1922, Oct. 23, Vol. 175, No. 17, pp. 734-736.
<i>Spermophilus guttatus</i> .	Ural Govt.	Zentral. f. Bakt. 1912, Vol. 65, No. 4-5, pp. 243-256.

- Remarks :
- Preliminary experiments were made by Strong (Mukden Conf. Rep. p. 239) and by Shibayama (ibidem p. 31).
  - Hamsters were also found susceptible in the Kirghiz Steppes (Tartakovski, Vrach 1900, No. 33).
  - "Gophers are not sufficiently susceptible to infection with B.P. to be of any importance from an epidemiological point of view." (McCoy, l.c.).
  - P. 23 of same report states: "In Komombo, however, *M. norvegicus* and *Arvicanthis niloticus* are the rodents which convey the infection to man."
  - "Field and forest mice" were found susceptible by Nuttal, (Centralblatt f. Bakt., XXII/4).
  - Experiments with the Ural tarabagan were carried out 1903 by Shurupoff (Russki Vrach 1911, No. 33, pp. 1301-1306). Flu (Geneesk. Tijdschr. v. Nederl. Indie, 1914, No. 5, pp. 540-551) performed exp. with marmots (?).



TABLE III. RODENTS OF TRANSBAIKALIA.

Name.	Remarks.	Parasites.
1) Tarabagan <i>Arctomys bobac</i> (Schreb)		<i>Flea</i> : Ceratophyllus Silantiewi Wagner 1898. <i>Louse</i> : Haematospinus lyrio- <i>Tick</i> : Rhipicephalus (? R. cephalus Burmeister. haemaphysaloxides). <i>Intestinal</i> : Ascaris sp.
2) Suslik <i>Spermophilus</i>		
a) ? Sp. Eversmanni Brandt.	a) Big var. with mottled back, found in the steppe near forests.	<i>Flea</i> : Ceratophyllus tes- quorum Wagner 1898. <i>Lice</i> : Genus Polyplax. <i>Tick</i> : Not determined.
b) Sp. dahuricus.	b) Small var. with uniformly coloured back, living in the open steppe.	
3) Striped squirrel <i>Tamias striatus</i> .	Found on the borders of the steppe where the first birch groves begin.	
4) Common flying squirrel <i>Pteromys sciorepteus</i> <i>volans</i> .	Found on the borders of the steppe where the first birch groves begin.	
5) Squirrel <i>Sciurus</i> .	Found on the borders of the steppe where the first birch groves begin.	
6) Domestic mouse <i>Mus musculus</i> .		<i>Fleas</i> : Ctenopsylla musculi Ceratophyllus fasc. <i>Mites</i> : Genus Laelaps, fam. Gamasidae.
7) Rats <i>Mus norvegicus</i> .		

(Continued on next Page.)

TABLE III.—(Continued).

Name.	Remarks.	Parasites.
8) Hamster		
a) Streaked hamster <i>Cricetulus grisens</i> A. N. Edw.		Leptopsylla (Ctenopsylla) pectiniceps.
b) Ordinary one-coloured <i>Cricetulus furunculus</i> .		Neopsylla compar. Rothsch. 1911 Ceratophyllus spec.
c) ? <i>Cr. rongarus</i> Pall. Variety of 8/b or with pointed ? <i>Cr. obscurus</i> snout and darker colour found A. N. Edw. near rivers.		Ceratophyllus spec.
? d) <i>S. dipoda</i> .	Very small variety found only round marshes & lakes near the ground water.	
9) Jumping hare <i>Alastaga mongolica</i> Radde.		Lice : Species?
10) Rat hare <i>Ochotoma dahurica</i> Pall.		Four species Ceratophyllus spec. Mites : Genus Laelaps, fam. Gamasidae.

TABLE V. TARABAGANS WITH NATURAL PLAGUE.

No.	Date found.	Locality.	Remarks.
1.	Autumn 1907.	Bet. Lake Dalai-nor and Man-chouli.	No buboes, lungs free. Suspicious chronic P.
2.	June 17, 1911.	Lake Charbada	Decomposed. Rt. axill. bubo. Smears and cultures pos. Exps. negative.
3.	June 24, 1911.	Sharasun	Cerv. buboes. Haem. in lungs. Exp. upon tarab. positive.
4.	June 26, 1911.	Arabulak	No buboes, lungs free. Exp. upon mice pos.
5.	Sept. 16, 1921.	Kinkija (5 v. from Suktui)	Cerv. and axill. buboes. Haem. lungs. Gp. experim. pos.

(Continued on next Page.)



TABLE V.—(Continued.)

No.	Date found.	Locality.	Remarks.
6.	Sept. 19, 1921.	Kinkija (5 v. from Sektui)	Inguin. buboes. Rest „
7.	Sept. 1921.	Kinkija (5 v. from Sektui)	Remnant only. Smears from muscle pos.
8.	Sept. 22, 1921.	Barun Zasulan (10 v. from Sektui)	Cerv. bubo. Smears pos. •
9.	Apr. 29, 1923.	Chistokina (1½ v. from Sektui)	No buboes. Lungs congested. Smears pos. Cultures and exps. neg. Dissected two days after found.
10.	June 4, 1923.	Barun Zasulan	Decomposed. Left cerv. bubo. All tests pos.
11.	June 8, 1923.	„	No buboes. Haem. foci in lung. Nodes in spleen. All tests positive.
12.	June 9, 1923.	„	Cerv. buboes. Haem. foci in lungs. All tests pos.
13.	June 23, 1923.	„	Decomposed. Left axill. and left cerv. buboes. Pneum. areas in lungs. Gp. exp. pos.
14	July 4, 1923.	Substn. 82.	Remnant only. Smears from muscle pos.
15.	July 21, 1923.	Sun Zasulan	„ „
16.	July 25, 1923.	Barun Zasulan	Complete specimen. Harbin Museum. Cerv. buboes. Pneum. areas lungs. All tests pos.
17.	Aug. 24, 1923.	„	Cervical glands affected. Many pneumonic areas lungs. All tests positive.
18.	Sept. 1, 1923.	Near Dauria	Cervical bubo. Nodes in lungs. Many nodes in liver. All tests positive.

It will be seen from above that the naturally infected tarabagans were mostly confined to an area around Sektui. The plague detachment worked about this district because of its regular periodic outbreaks. It is quite possible that other foci might have been located if the same attention had been paid to them, but difficulties of communication and transportation have to be considered.

TABLE VIII. WINTER EXPERIMENTS 1922-23.

No. of tarabagan	Date inf.	Mode	Condition during experiment	Date death	Days sick	P. M. Result.
T. 197	9/XI	Inhaled	Continuously feverish	16/XII	37	Chronic Plague.
T. 223	27/XI 14/XII	„	Hibernating	25/II	73	Smears suspicious, but cultures sterile.
T. 233	21/II	„	„	14/III	21	Acute Plague.
T. 238	21/II	„	„	3/IV	41	Resolving Plague?
T. 231	8/III	„	Feverish	13/III	5	Acute Plague
T. 232	8/III	„	„	14/III	6	„ „
T. 224	19/II	Nostrils	Hibernating	26/II	7	„ „
T. 250	1/III	„	„	26/V	86	Negative for Plague.
T. 241	30/I	Cutan.	„	14/IV	74	Resolving Plague?
T. 236	12/III	Conj.	Slight local react. Feverish	29/III	17	Subacute Plague.
T. 251	2/III	„	Sick, not hibernating	28/V	87	No signs of Plague.
T. 213	11/XI	Contact	Hibernating			No signs of Plague. Fed.
				12/I	62	corpse 197 16/XII!!
T. 234	Contact from 26/XI-14/III fed corpse 232		Hibernating	19/III	113	Acute Plague (Intestine affected).





Three tarabagans in the hibernating stage, Feb. 1923.

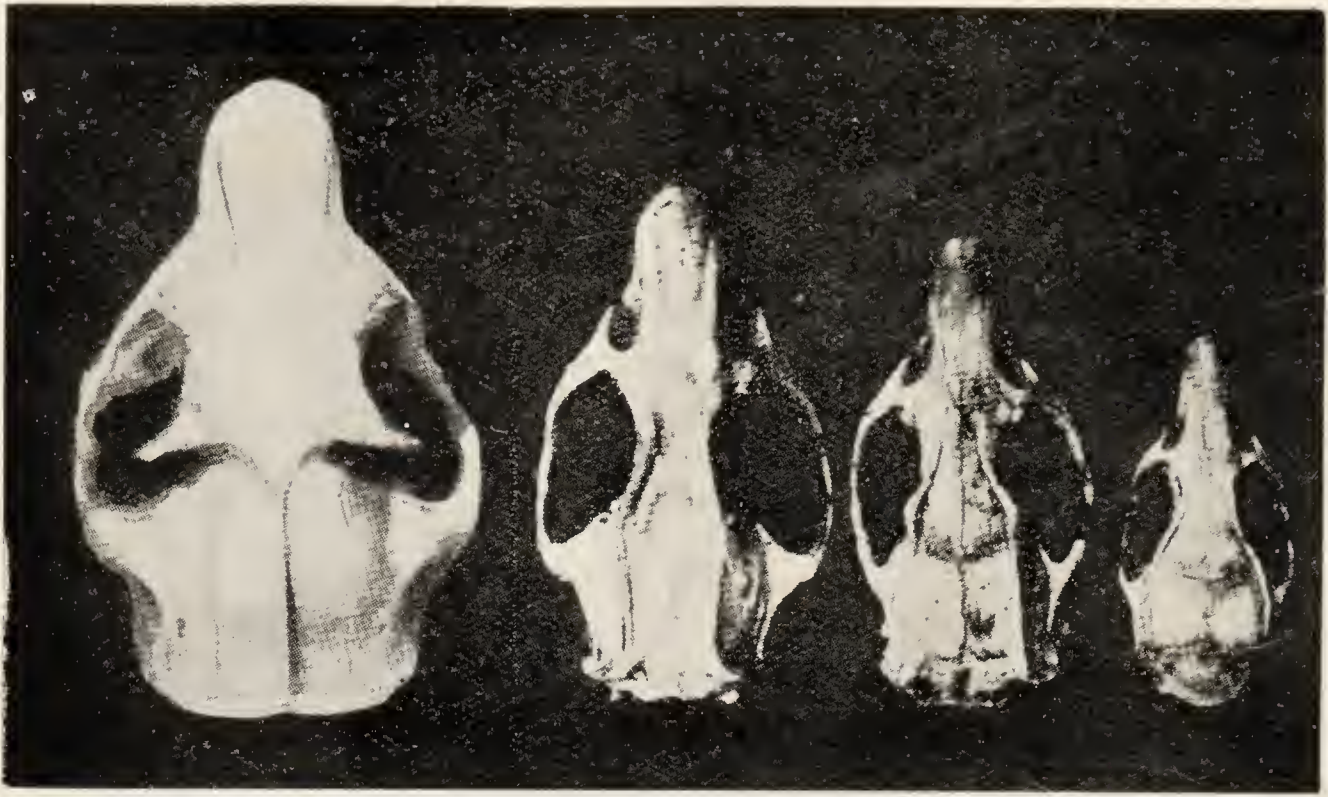
月二十民狀期眠冬在獭旱三



A plague infected tarabagan during hibernation held in gloved hands, Feb. 1923.

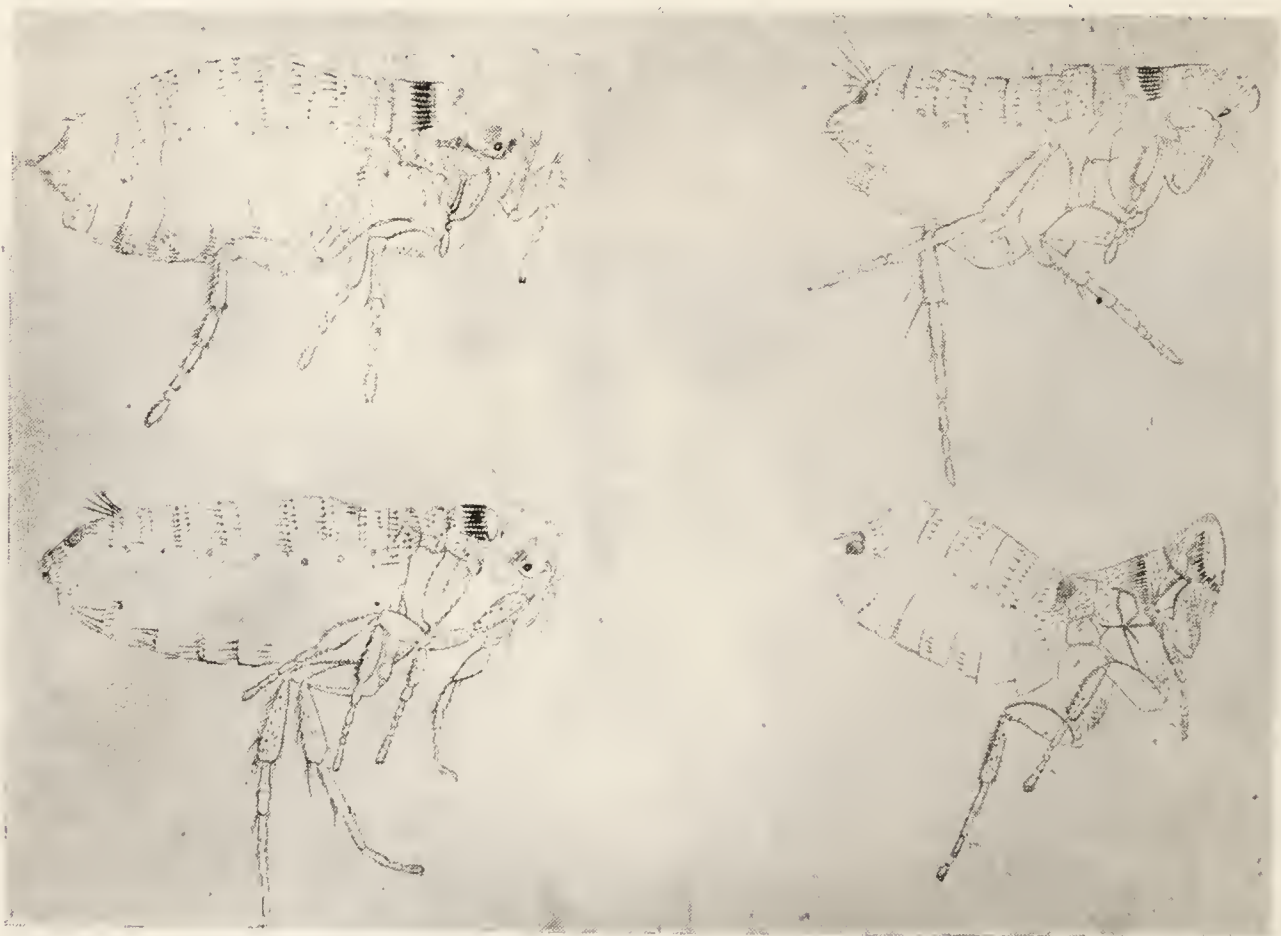
月二十民獭旱眠冬疫染住持手套皮戴用





Comparative sizes of skulls of different rodents. From left to right: *Arctomys bobac* (tarabagan), *Gunomys*, *Mus decumanus* and *Mus rattus*.

各齧獸類大小比較由左至右旱獭根羅埋屬大鼯家鼠



Fleas of some wild rodents found in Transbaikalia.

在薦士蘭加搜羅野齧獸之蚤類

- I. Tarabagan Flea, *Ceratophyllus*  
Silantievi Wagner 1898, M.
- II. Suslik Flea (*Spermophilus*  
*Dahuricus*), *Ceratophyllus*  
Tesquorum Wagner 1898, F.

- III. Rat-hare Flea (*Ochotoma*  
*Dahurica*), *Ceratophyllus*  
Spec., F.
- IV. Hamster Flea (*Cricetulus*  
*Griseus*), *Ctenopsylla* *Pecti-*  
*niceps*, F.





Railway wagons in Transbaikalia used by the Russo-Chinese Plague Expedition in June, 1923.

輻車用所加拜士蘭薦在隊究研疫鼠俄中月六二十民



Dissecting tarabagans in the open air. Russo-Chinese Plague Exped. 1923.

獾旱剖解天露在隊究研疫鼠俄中二十民



New Quarantine Camp, Newchwang, completed 1924.

成落年三十國民所離隔新莊牛





TABLE IX. FLEAS OF RODENTS SUFFERING FROM NATURAL  
PLAGUE.

<i>Host :</i>	<i>Flea :</i>
Tarabagan. <i>Arctomys bobac.</i>	Ceratophyllus Silantievi. Wagner 1898.
Californian Ground squirrel. <i>Citellus beechyi.</i>	Ceratophyllus acutus Baker Hoplopsyllus anomalus Baker.
Gerbille, <i>Tatera lobengulae.</i>	Dinopsyllus lypusus Xenopsylla eridos Listropsylla stygius
Multimammate mouse <i>Rattus coucha</i>	Dinopsyllus lypusus Echidnophaga larina
Striped mouse <i>Arvicanthis pumilio</i>	Dinopsyllus lypusus Xenopsylla eridos Chiastopsylla octavii Lystropsylla stygius
<i>Cricetomys gambianus</i>	Pulex cheopis (R) Ctenophalus serraticeps
<i>Pelomys fallax iredescenes</i> <i>Mus (Thamnomys) aff. dolichurus</i>	Loemopsylla cheopis Rarely : Ctenopsylla musculi and Ceratophyllus fasciatus.
Squirrel <i>Sciurus palmarum</i>	Fleas similar to Ceratophyllus fasc. Loemopsylla cheopis
<i>Nesokia bengalensis</i>	Pulex cheopis
Rabbit <i>Lepus cuniculus</i>	Spinopsyllus cuniculi Ceratophyllus fasciatus
Hare <i>Lepus timidus</i>	Spinopsyllus cuniculi
African bush-rat <i>Golunda campane</i>	Xenopsylla cheopis.

# A STUDY OF THE MORBID HISTOLOGY OF THE 1921 MANCHURIAN PLAGUE EPIDEMIC.

BY

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WITH 5 COLORED PLATES AND 7 MICROPHOTOGRAPHS.

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| 4. Bronchus.   | 13. Suprarenal Gland.         |
| 5. Tonsils.  | 14. Thymus.                   |
| 6. Uvula, Tongue.                                    | 15. Kidney.                   |
| 7. Oesophagus, Stomach.                              | 16. Heart.                    |
| 8. Uterus, Ovary, Tubes.                             | 17. Spleen.                   |
| 9. Testes.   | 18. Liver.                    |
| 19. Lung, Bronchioli, Pleura.                        |                               |
| 20. Lymph Glands (mesenterial, cervical, bronchial). |                               |
| 21. Discussion and Conclusions.                      |                               |
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### 1. Introduction.

The following descriptions are based upon the examination of small specimens from twenty three cases of pneumonic plague of the epidemic of 1921 in North Manchuria supplied by Dr. Wu Lien Teh. As it has not been possible to give a detailed description of every case, the following is a summary of the pathological changes found in the different organs as represented by the specimens which have been examined. Slides have been made in the usual manner and several stains have been attempted with each specimen to make possible an examination of the histological changes, the elastic fibres, "gitterfaser," fibrin and also the bacteria. The latter are stained by Loeffler's methylene blue, azure II, Giemsa, Romanowsky, etc. For the purpose of control Gram's staining was used.



In view of our studies of the pathological anatomy of pneumonic plague during the epidemic of 1911 in South Manchuria we have herein incorporated some of the results of the investigations of this disease which were made at that time. The illustrations accompanying this report are from the epidemic of 1911, but they accurately represent the conditions that exist in these specimens and are herewith published for the first time.

2. *Larynx*. One case.

No severe histological changes, except a considerable hyperaemia in the submucous tissue. Some round-cell infiltration was noted. Both hyperaemia and round cell infiltration occurred in and about the mucous glands.

3. *Trachea*. Seven cases.

In some cases the epithelial cells of the mucous membrane seemed to be but slightly affected, if at all. In others, however, desquamation, necrosis or destruction of epithelial cells were occasionally noted, but without serious ulceration. At the points where the epithelial cells were destroyed plague bacilli were found in the surrounding tissue. In one case there was a dense accumulation of bacilli in the sub-epithelial tissue in the neighborhood of a mucous gland. The changes in the mucous membrane varied in different cases and in location. Without exception, however, a marked hyperaemia of the sub-mucous tissue was noted, and this was often accompanied by round-cell infiltration. The outer walls appeared to remain unaffected and to show no evidence of hyperaemia or cell infiltration.

4. *Bronchus*. Five cases.

The condition of the wall is similar to that of the trachea. In addition, small local haemorrhages occurred in the mucous membrane, which penetrated into the subepithelial tissue. The inner surface of the wall was covered with detritus, blood, or desquamated and destroyed epithelial cells. A high degree of hyperaemia was noted in the submucous tissue, as well as some round-cell infiltration. Blood capillaries in the mucous glands were dilated and a mucous condition marked many of the gland cells. Plague bacilli were abundant in the mucous membrane and sub-mucous tissue. In some cases these even penetrated as far as the mucous glands. Where the augmentation of the bacilli in the mucous membrane was very dense, the covering epithelial cells were destroyed. The histological changes noted

in these specimens are found to be less marked than in the cases that were studied in the plague of 1911, but the general conditions are very much the same.

5. *Tonsils.* Eight cases.

Marked hyperaemia in all cases. The Tonsils were more or less rich in parenchymatous cells. In some cases the so-called "germinating centers" appeared greatly enlarged. These "germinating centers" were composed of large mononuclear light cells, including histiocytes which act as phagocytes. These histiocytes containing plague bacilli were also frequently observed in other parts of the glands. It is noteworthy that in some cases these enlarged "germinating centers" were not to be found.

A slight inter-cellular deposition of hyaline substance was occasionally observed.

The epithelial layer covering the surface of the tonsil appeared in most parts to be generally intact, or at least not destroyed. Here and there, however, destruction of epithelium took place, accompanied by destruction or necrosis of tonsillar cells of the superficial layer, and an emigration of leucocytes. More or less of inflammatory exudation might have occurred but no considerable destruction of tissue was noted.

Plague bacilli, more or less isolated or in masses, existed in the cavities of the surface and also within the tissue. The penetration of the bacilli into the tissue at the points of epithelial destruction was dense and the bacilli were found to be massed about the lymph follicles. Besides the plague bacilli numerous Gram-positive bacteria were also observed, especially in the cavities of the surface, and occasionally in small numbers within the tissue.

While these tonsils showed considerable hyperaemia and some necrosis of the cells with inflammatory exudation, the pathological changes were not particularly marked. In comparison with the changes that occur in the lungs and neighboring lymph glands they are very slight. This condition agrees with that observed in the epidemic of 1911, with one exception. In that case, the lymph glands of the neck showed such remarkable enlargement with haemorrhages as to indicate that the primary infection had occurred in these glands rather than in the lungs. Also the tonsils and surrounding tissues of the pharynx and larynx suffered great pathological changes. None of the present specimens exhibited any such changes as occurred in this particular case.



6. *Uvula*. Five cases. *Tongue*. Seven cases.

These specimens revealed only very slight pathological changes, although considerable hyperaemia had occurred. The epithelial layer was intact, or at least showed no marked evidence of destruction. In some cases subepithelial round-cell infiltration had taken place.

7. *Oesophagus*. One case.

No special histological changes in the tissue. There was, however, a marked dilatation and swelling of the small veins. The epithelial layer remained intact without evidence of haemorrhage. Within the submucous tissue small colonies of plague bacilli were noted in the clefts of the tissue which were unaccompanied by inflammatory changes. These colonies of bacilli were probably the result of postmortal propagation.

*Stomach*. One case.

No marked changes in the tissue of the mucous membrane. The epithelium remained intact and showed no signs of mucous obstruction. The veins and capillaries of the mucous membrane and submucous tissue indicated a condition of hyperaemia. No plague bacilli were found in this specimen, but whether this was due to their absence or to poor staining is not clear.

8. *Uterus, Ovary, Tubes*. Two cases each.

No marked pathological changes. A puerperal condition existed in one case and the tissue of the uterus showed characteristic tissue changes, but there was no evidence of plague infection. Plague bacilli were noted in the blood vessels only and were not numerous.

9. *Testes*. Two cases.

Tissue changes characteristic of the age of the patient were noted without evidence of pathological changes in the parenchyma or interstitium. The formation of spermatozoa did not seem to be materially affected. Because of inability to perfect the staining of the plague bacilli in the blood it was impossible to determine whether they had been able to enter the lumen of the seminal tubule through the apparently intact wall.

10. *Placenta*. One case.

It is not evident that any special structural changes had been caused by the plague infection. No plague bacilli were noted in the blood vessels of the chorion-villus. In some

chorion-villi hyaline-necrotic changes were observed, but these were not necessarily pathognomic of plague.

*Umbilical Cord.* One case.

No special tissue changes; no plague bacilli were stained.

11. *Brain.* Two cases.

Some postmortal and other artificial changes were noted. Where complete fixation had occurred the nerve cells showed no structural changes. The nuclei of most of the cells appeared intact, but the condition of the Nissle's corpuscles could not be ascertained. There was no evidence of cell infiltration, and haemorrhage occurred only slightly in the pia. Plague bacilli were observed in the blood vessels only, being most numerous in those of the meninges.

12. *Pancreas.* One case.

Plague bacilli only in small numbers in the blood vessels. No marked changes in the tissue of the parenchyma, interstitium or in Langerhans's islands.

13. *Suprarenal Gland.* Two cases.

No structural changes were observed. No marked changes in the "Gitterfaser." Hyperaemia occurred in one case. The cells of the cortical layer suffered no necrotic changes. Contents of lipoid not marked. A slight haemorrhage in the zona reticulus was noted in one case. The medullary layer was well preserved, no destruction or hypertrophy being apparent.

14. *Thymus.* Five cases.

No special structural changes. Plague bacilli were observed in large numbers in a few cases in the blood vessels and capillaries. Isolated bacilli might have been scattered about within the tissue, but no accumulations were found. No necrosis, haemorrhage or inflammatory areas were observed. Hyperaemia evident in most cases.

15. *Kidney.* Ten cases.

Hyperaemia to some extent in every case. While the exact condition of some specimens could not be determined because of postmortal changes, in those which had been properly fixed some cloudy swelling of the parenchyma was noted. Though the specimens were not suitable for fat coloration, numerous fine red granules or droplets stained by Sudan III were found, mainly in the epithelium of the convoluted tubules and also in the limbs of Henle's loop-tubules. While the presence of fat granules does not necessarily indicate



degeneration of cells, parenchyma cells which undergo cloudy swelling tend toward slight fatty degeneration. No extensive necrosis, fatty degeneration or destruction of parenchym cells was noted, although in a very few cells a lack of staining of nuclei was evident. The lumina of the tubules in some cases contained hyaline globular substance. The presence of a very small number of hyaline cylinders was noted in a few cases.

The fixation of the tissue did not allow for a satisfactory staining of Altmann's granules. These seemed to be quite irregular and diffused. This change occurs in case of plague, and while it is impossible to reach an exact conclusion on this point in connection with these specimens, it is probable that this condition exists.

Important changes were noted in the glomeruli; hyaline thickening of the capillary walls of the glomeruli was often evident. This condition was also often observed in the cases during the plague of 1911. While it has not been possible to discover any well marked hyaline fibrous thrombi in the capillaries of the glomeruli, as have been mentioned by some authorities, this does not indicate that they are absent here.

In no case was haemorrhage into the cavity of the Bowman's capsules or the urinary tubules observed. This agrees with the results of investigations made in the epidemic of 1911. Such haemorrhage, if it occurs, must be rather unusual. Only in one case was any cloudy substance noted in the cavity of Bowman's capsules. Also in one other case there was observed somewhat of an accumulation of polynuclear leucocytes in one glomerulus. On the whole, it is apparent that the glomeruli are affected by plague virus.

Plague bacilli in the blood vessels of the kidney and also in the capillaries of the glomeruli were always seen. This was not the case, however, in the lumen of the tubules. In one of these cases a very few isolated Gram-negative bacteria were noted, the morphology of which appeared similar to those of plague bacilli, but it was not possible to identify them as such. In the epidemic of 1911 bacilli were found within the rectus tubules in only one case. While the passage of plague bacilli into the urinary tubules may not be impossible, this is not in any sense a usual or necessary feature. The relatively small number of plague bacilli in the blood vessels of the kidney as compared with those in the liver corresponds to our observations in the epidemic of 1911.

#### 16. *Heart.* Five cases.

In no case was there any marked change in the structure and arrangement of the muscular fibres. In general the form

and staining of the nuclei indicated that they were intact. On the whole the striation of the muscular fibres was distinct, but in some cases this showed signs of being disturbed. No marked degenerative changes were observed. Because the fixation of the specimens was not suitable for fat staining, Sudan III did not produce good results. In specimens from the epidemic of 1911 Sudan III revealed fine fatty globules in the muscular fibres. However, so far as these specimen are concerned, the muscular fibres in places were somewhat marked by cloudy swelling and it is possible that they might have undergone some fatty degeneration. No special cell infiltration in the interstitium, and no evidence of haemorrhage. His's fascicules remained unchanged. Because of the method of fixation the glycogen in these fascicules could not be stained. Plague bacilli were always present both in the larger blood vessels and in the capillaries.

17. *Spleen.* Thirteen cases.

More or less evidence of hyperaemia, the intensity of which varied in different parts of the organ. There were no indications of marked haemorrhage.

In general the pulp showed no marked cell proliferation, which in some cases was more or less hyperplastic. Eosinophile leucocytes and plasma cells were present only in small numbers. A very few giant cells of bone marrow were noted in one or two cases. Evidence of histiocytes or endothelial cells was often noted in the presence of comparatively large numbers of mononuclear cells containing much protoplasm. In some cases relatively numerous polynuclear leucocytes appeared, but in others they were not abnormally present. This condition was also noted in the epidemic of 1911.

No extensive necrosis of tissue or cells was apparent, except in one case, which exhibited a notable anaemic infarct of the spleen. Necrosis of small numbers of cells was observed here and there, however, especially in those localities where plague bacilli were densely accumulated. Often some structureless dirty hyaline substance was noted within the pulp. Such hyaline substance was located between the cells of the pulp or apparently along along the reticulum.

The lymph follicles of the spleen were generally small in size, sometimes abnormally so. None showed evidence of marked enlargement. This condition agrees also with investigations made in the epidemic of 1911. The follicles, which consist of lymphocytes, almost always showed clearly defined boundaries, though some exceptions were noted. While the tissue of most follicles remained apparently intact,



in some cases there were marked changes. Some deposition of hyaline substance within the follicle along the reticulum between the cells was noted. In one case the wall of the small artery within a follicle had undergone necrosis and in the neighboring area such hyaline deposition between the cells was apparent. In a few cases so called "germinating centers" containing enlarged and lightly stained cells were noted. Some of these were certainly histiocytes formed from reticulo-endothelial cells and acted as phagocytes for the disposal of broken-down cell substance. Some measure of hyperaemia surrounded the follicles.

Deposition of subendothelial hyaline substance was noted in the walls of the small arteries. In one case the "Gitterfaser" of the spleen was stained but this showed no evidence of special thickening or diminution.

Plague bacilli were found to be abundant in the blood space of the pulp. Between the pulp cells they appeared either singly or in colonies. They were mostly free between the cells but a few were phagocyted. Regarding the phagocytosis of plague bacilli in the spleen the specimens were not suitable for minute examination, and this report must accordingly remain incomplete. But the histological examination of the epidemic of 1911 showed phagocytes originating from endothelial or reticulo-endothelial cells or histiocytes in the pulp. The same condition must have happened in these specimens also.

The lymph follicles were found to contain plague bacilli, but they were less numerous than in the pulp. The bacilli were mainly limited to the surrounding or peripheral zones of the follicles, as very few were noted in the central parts. Gram-positive bacteria in the pulp were found only in small numbers in some cases.

#### 18. *Liver.* Fifteen cases.

The structure of the acini and cellular trabeculum seemed to be intact. No marked dissociation of the parenchyma tissue was apparent. More or less hyperaemia was evident. Some dilatation and fulness of the vena centralis and blood capillaries in the central parts of the acini were noted. The parenchyma cells, on the whole, appeared intact, although there was some evidence of cloudy swelling. The specimens were not suitable for fat staining, but Sudan III revealed in most cases more or less presence of fat droplets. The localization of these fat droplets could not be exactly determined, but in a number of cases these were most numerous in the central parts of the acini.

In general no marked cell necrosis was found, but in some cases the nuclei of the epithelial cells did not respond well to the stain. No inflammatory cell infiltration was anywhere noted; nor were any metastatic suppurative areas, such as those reported by some authorities in the liver in cases of bubonic plague, apparent. Except in the case of a foetus of a plague mother, there was no augmentation of lymphoid cells of lymph follicle-like character, but the presence of these lymphoid nodules might not have been especially abnormal in the foetus.

No signs of haemorrhage were to be found. Plague bacilli were observed in the blood vessels and capillaries in fairly large numbers. Phagocytosis of the bacilli by endothelial cells was taking place. Gram-positive bacteria were found in the blood capillaries in small numbers in only a few cases.

19. *Lung.* Fifteen cases.

Pneumonic plague causes most important and very complicated tissue changes in this organ. Several stages of histological change, from simple hyperaemia, or hyperaemia with slight serous exudation, to severe cellular exudation, were observed. More or less haemorrhage occurred in various parts of the lungs. Haemorrhage within the alveoli in these cases was not especially severe, but erythrocytes in larger or smaller numbers within the alveoli were always apparent. In most cases, in and under the pleura, haemorrhage was histologically very marked. Some haemorrhage was also noted in the interstitium, especially in the interlobular septum and within the connective tissue surrounding the bronchi and blood vessels. At the root of the lung in the connective tissue surrounding the bronchi and blood vessels severe haemorrhage was sometimes observed.

Fulness and dilatation of the capillaries of the alveolar walls was noted even in portions of the lung where no plague bacilli were found in the alveoli, or no inflammatory exudation had taken place. This hyperaemia was accompanied by serous exudate which filled the alveoli and contained a few exudative cells, especially leucocytes. In the early stages of inflammation, plague bacilli occurred within the serous exudate in fairly large numbers. Where the inflammation was more advanced the alveoli became filled with cells, polynuclear cells being most numerous, but which were accompanied by mononuclear cells, the latter being mostly histiocytes and desquamated epithelial cells. These cells in the exudate were more or less mixed with erythrocytes and serous exudate which contained a comparatively very small quantity of fibrin.



The degree of density of cells in the exudate within the alveoli varied greatly; in some cases the cells were loosely arranged while in others they were closely packed together. In every case the alveoli contained dense masses of plague bacilli. In no other form of pneumonia are the pathogenic bacilli to be found in such abundance. It is also characteristic of plague pneumonia, as compared with ordinary pneumonia, that fibrin exists within the exudate only in small quantities. The other important histological characteristic of plague pneumonia is the frequent appearance of hyaline substance in the alveolar walls. The presence of this hyaline material is accompanied by various changes in the walls of the alveoli. Where these changes occur the latter are irregularly thickened. This enlargement of the alveolar walls in one case was due to a hyaline thickening of the capillary walls, in another it was due apparently to hyaline thrombi within the capillaries, and in still other cases to a hyaline deposition on the outside of the capillary walls or within and inside of the alveoli walls themselves. This hyaline deposition was also observed in the epidemic of 1911. At that time, in addition to a deposition on the alveolar walls, a similar hyaline substance was observed to radiate out from the walls of blood vessels. In the present specimens a somewhat similar deposition seemed to have occurred, but the radiations were not especially marked.

The character of this hyaline substance is difficult to determine and in chemical composition it may not be identical; a part of it without doubt is composed of fibrin. In one case this hyaline substance within the lumen as well as on the walls of the blood capillaries of a number of alveoli was very clearly stained by Weigert's fibrin-staining method, while the staining of the fibrin itself in the same specimen was not good. It is probable that this hyaline deposition is caused by the severe exudative inflammation of the lung and, excepting the fibrin thrombi, is a coagulated substance resulting from the exudation from the blood vessels. This is not necessarily a specific characteristic of plague pneumonia. The deposition of hyaline in and on the walls of the alveoli also occurs, for instance, in influenza pneumonia. Giant cells of bone marrow are often to be found in the blood capillaries of the lung tissue.

"Gitterfaser" and elastic fibre showed no distinct changes in those portions of the lung which were not affected by inflammatory exudation. Where marked exudation did occur, however, some diminution and even destruction of these fibres were to be noted.

*Bronchioli.*—Where a small area was affected by plague pneumonia and the inflammation was slight the epithelium



layer did not seem to be much affected. It frequently appeared to be regular in form and arrangement. In other cases desquamation and obstruction of the epithelial cells with mucus was present. Within areas affected by an advanced stage of pneumonia, the walls of the small bronchi were more severely affected, inflammatory infiltration of lymphocytes and polymuclear leucocytes occurred both in the walls and in the surrounding tissue, and was always accompanied by hyperaemia. The epithelium was largely desquamated or destroyed. A very interesting and important histological development was often noted in the small branches of the bronchi. Sometimes clusters of bacilli and cells of inflammatory infiltration were proceeding toward the lumen from the outside of the wall, the epithelium apparently still remaining intact. In these cases the destruction of the mucous membrane was being brought about by an invasion of the virus through the wall of the bronchioli rather than from within the lumen itself. On the other hand, at other points in the bronchioli destruction of the epithelium had occurred and small clusters of bacilli were noted in the subepithelial layer without evidence of marked changes in the surrounding wall tissue.

*Pleura.*—In those areas where the lung tissue showed no hepatisation the pleura appeared only slightly affected. Hyperaemia, however, was almost always seen. Also a thin lining of fibrous substance appeared to cover the surface of this portion of the pleura. The surface cells beneath this lining did not seem to be much affected, although necrosis occasionally occurred. The pleura covering a pneumonic area is usually much more severely affected. A more or less fibrous lining with many leucocytes appeared on the surface, while the cells beneath usually suffered destruction or necrosis. The small blood vessels on the inner side of the pleura showed exceptional hyperaemia. Haemorrhage in the pleura was very marked and formed a well defined layer beneath the epithelium. In addition there was often a pronounced infiltration of leucocytes into the subpleural and pleural tissue. Fibrin may also occur in very small quantities in these areas of pleuritis, but it is not especially marked. As will be mentioned later, plague bacilli are densely massed in the subpleural and pleural tissue. Occasionally at points on the pleura rather isolated colonies of plague bacilli accompanied by some leucocytes were noted. These patches did not appear to result from the general process of pneumonic hepatisation.

Investigation of the distribution of bacilli in the lung tissue is most important in studying the pathology of pneumonic plague. They are abundant in the alveoli within areas of hepatisation and also where the alveoli have discharged only



serous exudate. In the former case, where the alveoli are filled with cellular exudate, the bacilli are densely massed in the spaces between the cells. Of this great multitude of bacilli only a few seem to be phagocyted by the cells within the exudate. In some cases, the phagocytes for plague bacilli could only with difficulty be observed in the pneumonic area. This condition is also to be noted in the lumen of the bronchi. These cells which do act as phagocytes for the plague bacilli are mainly large mononuclear cells, most of which seem to be histiocytes. To what extent polynuclear leucocytes may act as phagocytes to the plague bacilli is difficult to determine.

While plague bacilli are to be found in abundance throughout pneumonic lung tissue, including the blood vessels and capillaries, the greatest accumulations occur in the lymph vessels and lymph spaces of the tissue. The lymph vessels at the root of the lung and those accompanying the branches of the bronchi and neighboring blood vessels are very often most densely packed with plague bacilli. The pathological change in the walls of the bronchioli, as noted above, where the destructive process was working through from the outside of the wall towards the mucous membrane within, was due to the virus being brought by the lymph vessels along the bronchioli.

It was peculiar of many of the blood vessels within the plague pneumonic area that their walls were surrounded by marked accumulations of bacilli. This may be due to some perivascular space which may provide passage for lymph and thus becomes densely filled with bacilli. Very often the wall of the blood vessel itself is invaded by the bacilli and if this is not too thick, as in the case of the veins, an accumulation of bacilli may penetrate to the interior where the endothelial cells are destroyed. In this way great numbers of plague bacilli enter the blood stream. These changes in the walls of the blood vessels were also noted in the epidemic of 1911. The lymph vessels, greatly dilated and filled with bacilli, contain also polynuclear leucocytes and lymphocytes and some blood.

While the bacilli found in greatest abundance in the pneumonic areas were almost entirely plague bacilli, mixed infection in varying degrees was not uncommon. In most of these specimens, gram-positive bacteria, such as diplococi, streptococci, staphylococci and other bacilli, were noted in the blood vessels and alveoli. The number of these bacteria, however, was much smaller than the plague bacilli. In a few cases the number of these other bacteria was fairly large in the pneumonic area, and they might have aggravated the pneumonic condition of the lung, but the chief source of pneumonic infiltration, whose manifestations were almost identical in all cases, is due to plague bacilli.

20. *Lymph Glands.* Mesenterial two cases, cervical two cases, bronchial five cases.

In the two cases of mesenterial lymph glands hyperplasia of cells accompanied by more or less hyperaemia was noted. In one of these a diffused hyperplasia of lymphoid cells had occurred, while the other case was marked by a proliferation of endothelial cells which had filled up the dilated lymph sinus. So called "germinating centers" in the follicles were not well developed. No necrosis, haemorrhage, or inflammatory areas were observed, and polynuclear leucocytes appeared only in small numbers. Bacilli were not numerous in the blood vessels. Examination of numerous slides revealed no plague bacilli in the tissue of the mesenterial lymph glands. In an occasional slide among the cells of the tissue a few bacilli were noted which resembled plague bacilli, but the exact character of the former could not be determined.

One of the two cervical lymph glands showed marked hyperaemia and proliferation of the endothelial cells of the dilated lymph sinus. In some the follicles "germinating centers" appeared. As to the existence of plague bacilli, because of poor staining, no definite results could be obtained. It is at least certain that no large accumulations of bacilli existed, and no tissue changes characteristic of plague were observed.

The other cervical lymph gland proved to be severely affected by chronic tuberculosis. A fibro-caseous substance occupied almost the whole gland, lymph tissue remaining only in one corner. This lymph tissue, especially within the lymph sinus, contained small accumulations of bacilli which resembled plague bacilli. Whether plague bacilli can penetrate into a tubercular area is an interesting question. So far as this specimen is concerned, no plague bacilli were found in the fibro-caseous substance.

The bronchial lymph glands, being most intimately connected with the lung, are the ones which undergo the severest histological changes in pneumonic plague. The entire tissue of the gland becomes heavily infected with plague bacilli. Where these were abundant, the cells of the gland were diminished in number. Cells undergoing necrosis were seen. No marked blood extravasation within the gland was observed. On the other hand the normal tissue structure of the lymph gland was severely affected. Elastic fibres and "Gitterfaser" were less stained and became attenuated or even more or less destroyed. Augmentation of bacilli occurred more markedly in the peripheral zone than in the central portions, and more in the lymph sinus than in the lymph follicles and medullary fasciculus. The presence of bacilli could be noted within the



follicles, where they were found scattered among the lymph cells, particularly if the gland was severely infected, but the lymph follicles were always less affected by the bacilli than other parts of the gland. Often follicles were observed to be surrounded by a layer of varying thickness consisting of a mass of bacilli. These seem to offer more resistance to the effects of plague virus than other parts of the gland tissue, but eventually they undergo a gradual atrophy and destruction.

Infiltration of polynuclear leucocytes into the tissue of the lymph gland occurred to some extent. Sometimes these appeared in fairly large numbers but no suppurative areas were noted. A limited amount of serous infiltration was occasionally observed and staining revealed the presence of fibrin, but only in small quantities. A deposition of hyaline substance on the walls of blood vessels and the reticulum, present in cases during the epidemic of 1911, may also have occurred, but it was not prominent. In spite of the presence of great numbers of bacilli and some augmentation of leucocytes and mononuclear migratory cells, phagocytosis in the lymph glands could not be clearly ascertained. In some cases no cells carrying plague bacilli could be found. But investigations during the epidemic of 1911 indicated that phagocytosis in the plague bubo did occur to some extent, especially in the early stages of the infection. This was noted particularly in the lymph sinus. In those cases the swollen and desquamated sinus cells or endothelial cells, which partook of the nature of histiocytes, acted to a large extent as phagocytes. Other mononuclear cells may also have become phagocytes. It was occasionally noted furthermore that blistered phagocytes which were carrying lymphocytes and blood pigment had also absorbed some plague bacilli. Where the augmentation of plague bacilli in the lymph sinus became very marked the swollen and desquamated cells and phagocytes disappeared. This is perhaps the reason why in the present specimens, which show advanced stages of plague bubo, phagocytosis is difficult to determine.

In those areas where carbon dust had been heavily deposited in the bronchial lymph glands the accumulation of plague bacilli was less marked.

The relation of the penetration of plague bacilli into tubercular areas of the bronchial lymph glands is the same as in the cervical gland mentioned above.

The tissue surrounding the bronchial lymph glands in the root of the lung was always infected with dense accumulations of plague bacilli, and severe haemorrhage was often noted. Lymph vessels near the bronchial glands appeared dilated and

filled with bacilli. Numerous leucocytes, lymphocytes, and some red blood cells were also found in these lymph vessels.

## 21. *Discussion and Conclusions.*

The most important tissue changes caused by pneumonic plague occur in the lungs and neighbouring bronchial lymph glands. In these organs the plague bacilli are most abundant and the tissue changes which take place are mainly due to the presence of these bacilli. The lungs are affected by hyperaemia, serous and cellular exudation and more or less haemorrhage. The specimens examined in connection with this report did not, however, reveal specially marked haemorrhage into the alveoli. Fibrin within the alveolar exudate was inconsiderable. Hyaline substance, the origin of which is more or less intimately related to that of fibrin, was in most cases found deposited on the alveolar walls.

These tissue changes, together with the enormous increase of plague bacilli in the pneumonic areas, are characteristic of plague pneumonia.

The plague bacilli are abundant not only in the alveoli but also in the interstitial tissue, especially in the lymph vessels and spaces in the immediate neighborhood of the bronchi and blood vessels and under the pleura. In these lymph passages the plague bacilli are usually found to be most densely massed. The lymph vessels at the root of the lung in the neighborhood of the infected bronchial lymph glands are likewise always greatly dilated and filled with bacilli. The lymph vessels seem to provide an especially favorable medium for the augmentation of the plague bacilli and at the same time they serve as convenient canals along which the latter can proceed to other parts of the tissue.

Examination of the lung shows small areas of plague pneumonia developing in the peribronchial alveoli. The multiplication of bacilli in the peribronchial and perivascular lymph vessels and spaces is a most important cause of this lobular pneumonic infection. On the other hand, the spreading of pneumonic areas may also be due to the inhalation of plague bacilli from the upper and wider parts of the bronchi and bronchioli into the deeper recesses of the lung. In this case the mucous membrane of the bronchioli is effected from within the lumen and the plague bacilli penetrate from the mucous membrane, whose epithelium is more or less destroyed, into the tissue of the walls. This inhalation of bacilli may also carry them directly into the alveoli.

The peribronchial and broncho-pneumonic areas whilst expanding join with each other, thus diffusing the pneumonic



infiltration through a large portion of the lung. When, however, the process of pneumonic infiltration occurs over a wide area in a very short space of time its relation to the condition of the bronchioli is not clear.

As to the condition of the cervical, mesenterial and bronchial lymph glands in pneumonic plague, it is the latter which are by far the most severely affected both as to pathological tissue change and the presence of bacilli which are especially augmented in the lymph sinus. The tissue of the mesenterial lymph glands showed only slight evidence of the presence of plague bacilli, and in the cervical glands no large accumulations of bacilli were found. Plague infection in an individual may result in some cellular hyperplasia in these lymph glands. The tonsils are only slightly affected, comparatively small accumulations of bacilli being found and these mainly in the superficial layer.

#### *Modes of Infection.*

During the International Plague Conference which was held in Mukden in 1911, two opinions were presented regarding the mode of plague infection. Those holding one opinion argued that the primary infection occurred in the tonsils and that the lungs were secondarily infected by virus carried by the blood. Others insisted that the virus was directly inhaled into the air passages. The writer (A.F.) supported the latter position at the Mukden Conference, and this present investigation offers no reasons for altering the opinion then set forth.

In no case in the above examination did the tissue changes in the tonsil or the accumulations of bacilli justify the presumption that this organ could be the seat of primary infection. On the other hand the lungs and neighboring lymph glands are always incomparably more severely affected and more densely infected by the plague bacilli. The tissue changes in the tonsils are more probably due to secondary infection by plague bacilli carried by the sputum. The histological condition of the tonsils as compared with the tissue changes in the bronchus and lungs leaves no doubt but that the primary infection follows upon the inhalation of the plague bacilli into the air passages. This does not mean, however, that the bacilli immediately reach the alveoli with the incoming breath. The inhaled bacilli may be deposited for a time on the some portion of the walls of the air passages, for instance at or near the bifurcation of the trachea. The multiplication and penetration of the bacilli may then occur in the walls and surrounding tissue. The lymph vessels will then be invaded and along these the multiplying bacilli will be carried to the neighboring

lymph glands and also toward the periphery of the bronchial system. Likewise, sooner or later, they may also be carried into the ductus thoraticus. At the same time the histological condition of the bronchus, as described above, which showed intensive augmentation of plague bacilli in the mucous membrane and submucous tissue, which are in direct contact with the air, indicates the possibility of the bacilli being spread through the bronchioli and alveoli by inhalation.

These are certainly the most important modes of infection. On the other hand, primary infection of the pharynx and tonsils, which may cause a cervical bubo, is, of course, not impossible; such a case was observed in the epidemic of 1911. In that case the tonsils were severely affected.

Without doubt the lungs and neighboring lymph glands are primarily infected by pneumonic plague, but secondary infection of these organs may, of course, occur as in the case of bubonic plague.

*Bacteraemia.* Bacteraemia is generally observed. The invasion of plague bacilli into the blood stream takes place through the ductus thoraticus which carries lymph from the infected areas into the veins. Also plague bacilli may directly enter the blood stream within the infected areas by breaking through the walls of the vessels, especially the veins, about and within which the bacilli may be accumulated. This penetration through destroyed portions of the walls of the veins is often observed in the lungs.

*Parenchymatous Organs.* The liver and kidney show some parenchymatous degeneration or cloudy swelling of the parenchyma which may incline towards fatty degeneration. (The specimens were not suitable for examination of fat.) Severe extensive degenerative changes of the cells is not apparent. Passage of plague bacilli into the urine does not usually occur. The glomeruli are more or less affected. The muscle cells of the heart are even less affected.

*Spleen.* The pulp of the spleen often contains considerable numbers of plague bacilli. The tissue changes of this organ consist of some proliferation of the pulp cells and endothelial cells in the earlier stages of the infection, some of which finally suffer necrosis. Blood circulation is disturbed. There is some inflammatory infiltration with emigration of polynuclear cells.

*Digestive Organ.* The tissue of these organs is not markedly affected by pneumonic plague.

*Hyperaemia.* In all organs more or less hyperaemia in small vessels and capillaries is noted and haemorrhage often



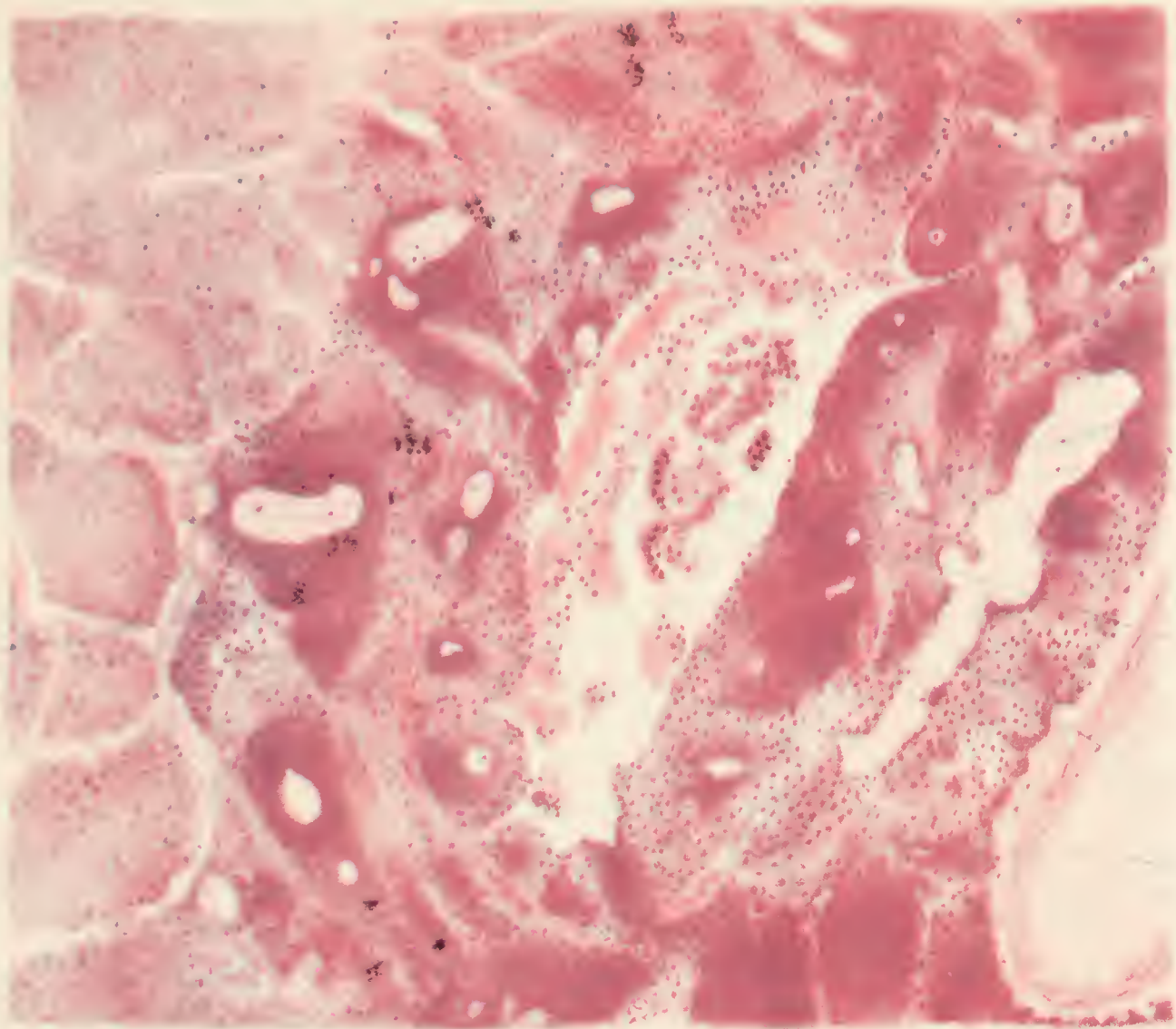


Plate 1. Section of bronchiole and lung in Plague Pneumonia. x 200 diam.  
Romanowsky stain.

- a. Groups of *B. pestis* around walls of bronchiole.
- b. Desquamation of epithelium of bronchiole.
- c. Some fibrous tissue also disintegrated.
- d. Congested blood vessels around wall of bronchiole.
- e. Alveoli of lung, mostly invaded by pneumonic process.

〔圖一〕 氣管枝一部及肺疫之肺藏二百倍大 魯免勞氏染色

- a. 疫菌圍繞氣管枝壁
- b. 氣管枝上皮細胞剝脫
- c. 結締組織示不完備
- d. 肺胞多被肺炎侵襲

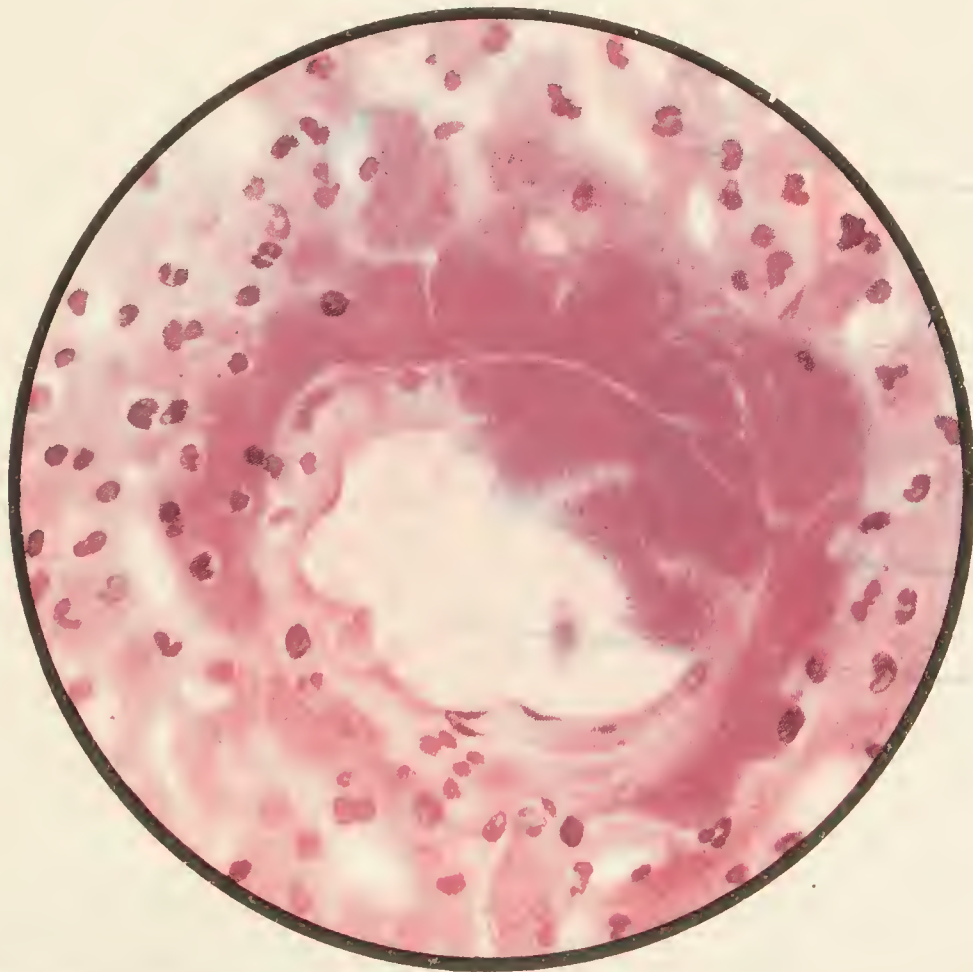


Plate 2. Section of lung in Plague Pneumonia. x 800 diam., oil immersion.  
Romanowsky stain.

- a. Masses of plague bacilli around small blood vessel.
- b. Large numbers of bacilli entering blood vessel.
- c. Congested blood vessel.
- d. Lung tissue.

〔圖二〕 肺臟內肺疫之一部

- a. 多數疫菌圍繞小血管
- b. 多數疫菌侵入血管內
- c. 血管充血
- d. 肺藏組織





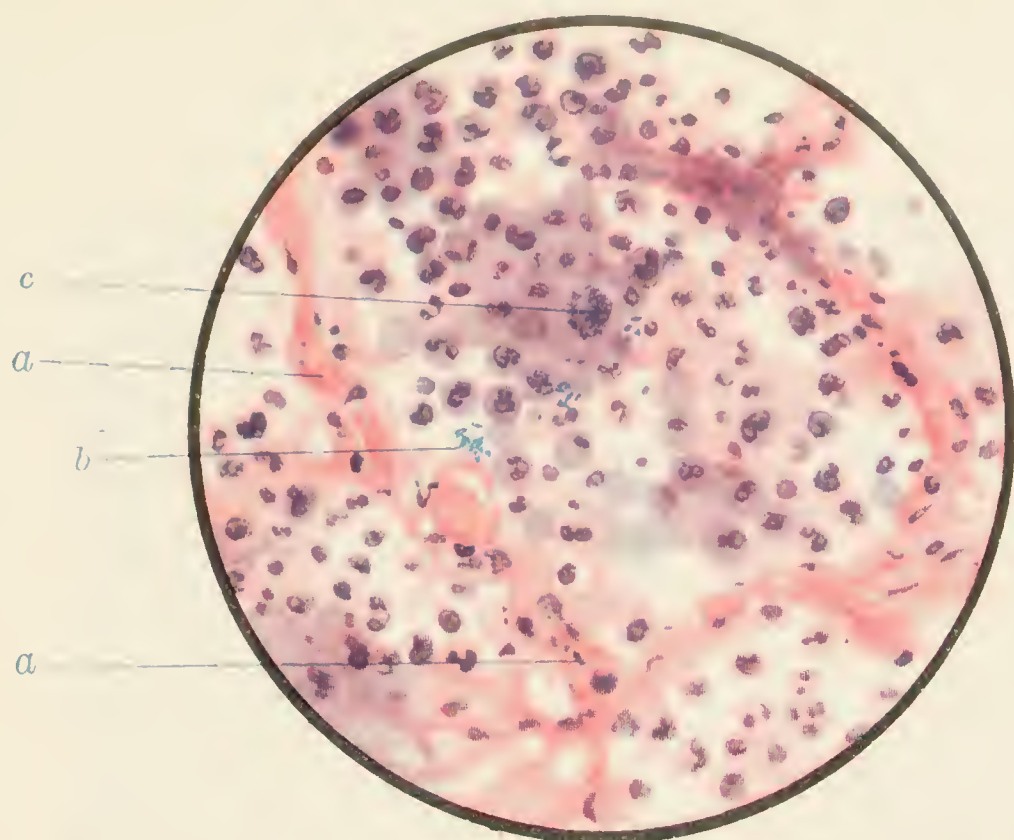


Plate 3. Section of lung in Plague Pneumonia. x 500 diam. Haematin and eosin stain.

- a. Hyaline deposits around alveoli of lung making this part thicker than normal.
- b. Plague bacilli among cells inside alveoli.
- c. Some bacilli seen inside leucocytes.

〔圖三〕 肺臟內肺疫之一部 五百倍歇麻田及依奧金染色

- a. 喜阿林變性圍繞肺胞使此部比平常增厚
- b. 疫菌在肺胞之細胞內
- c. 白血球內見疫菌

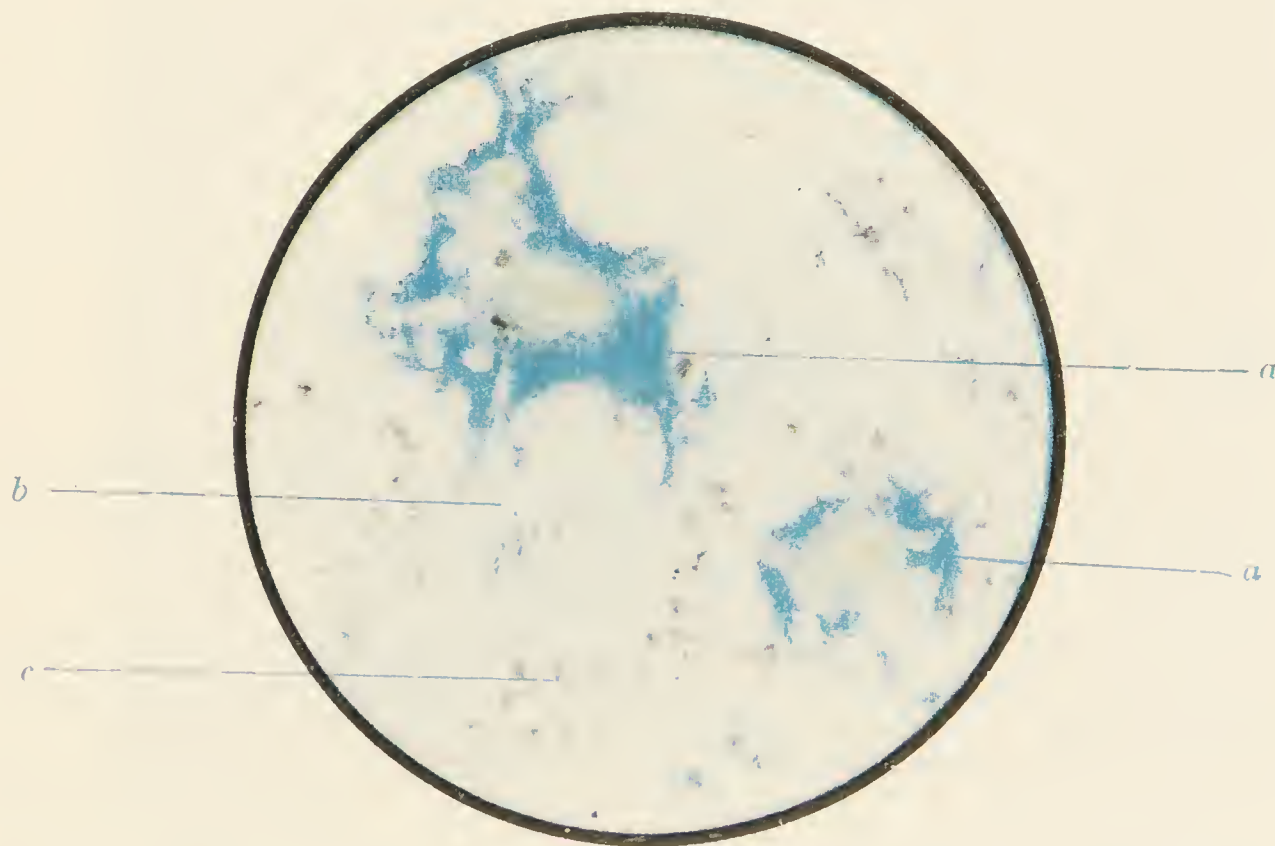


Plate 4. Section of lung in Plague Pneumonia. x 200 diam. Weigert stain.

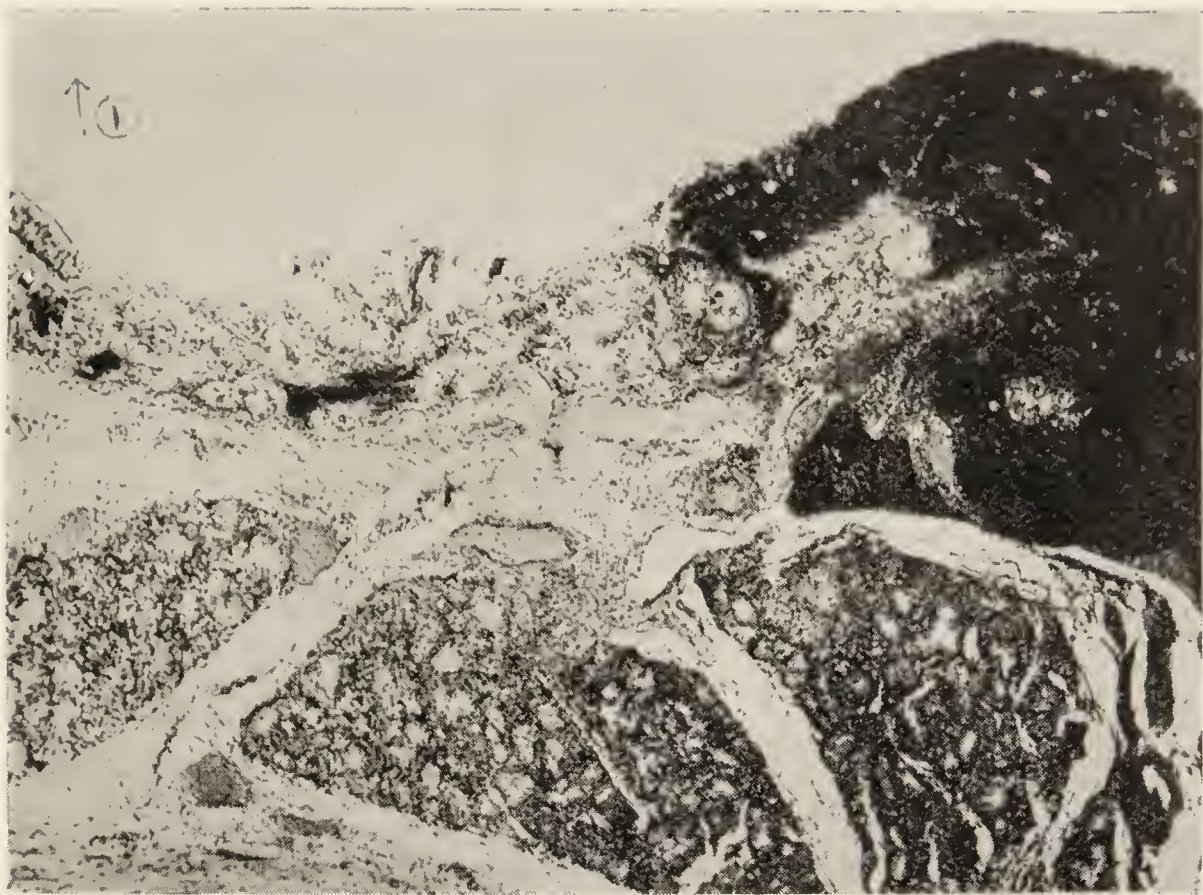
- a. Nets of fibrin around small blood vessel.
- b. Groups of plague bacilli in lung tissue.
- c. Lung tissue.

〔圖四〕 肺臟內肺疫之一部 二百倍維渣氏染色

- a. 纖維網圍繞小血管
- b. 在肺組織中之疫菌羣
- c. 肺臟組織



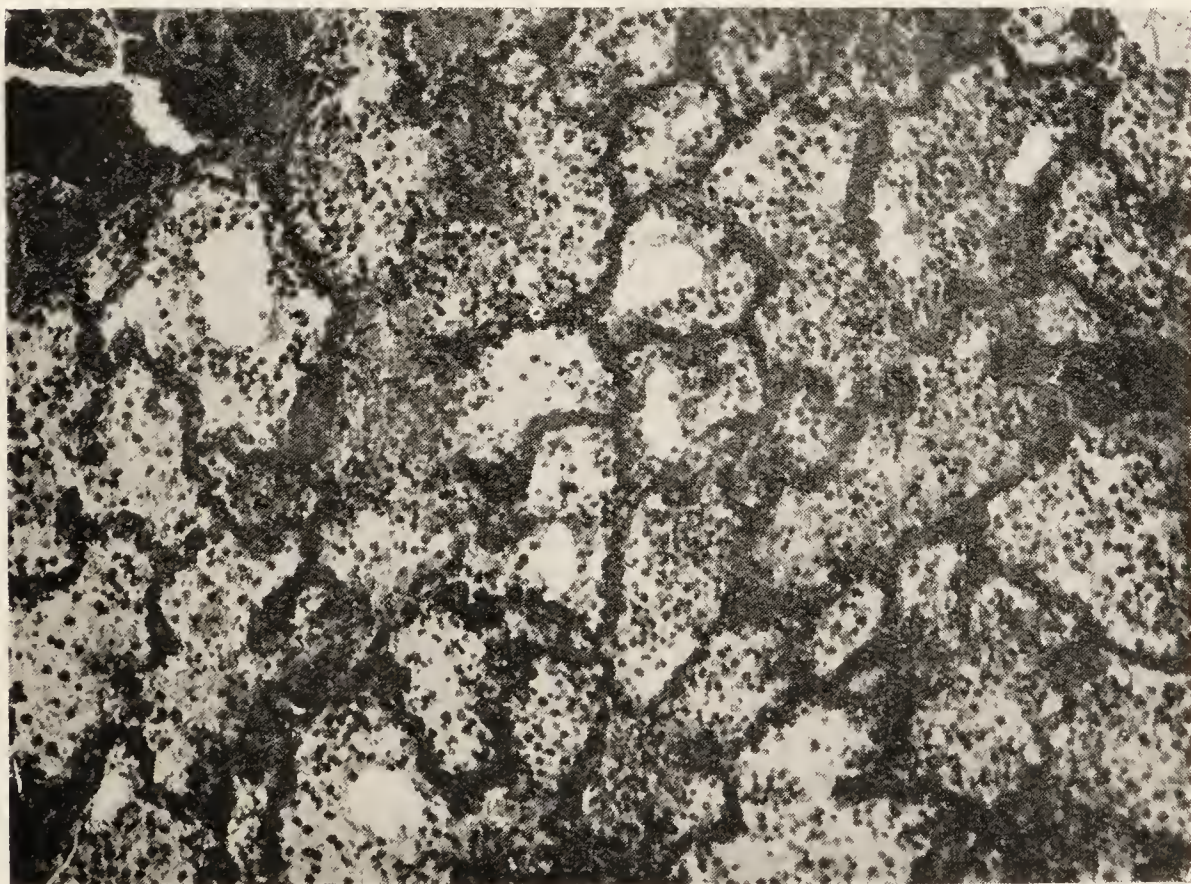




Section of lower part Bronchus (human), Plague Pneumonia, mod. magnified. On upper left hand corner note small portion of remaining epithelium of muc. memb. The black areas are dense masses of B.P. At right is a large agglomeration of B.P. proceeding from muc. memb. into the mucous glands.

(類人)部下管氣

菌疫示黑暗其部剩餘之胞細皮上膜粘有角上左疫肺  
內膜粘腺至起膜粘由羣大菌疫係部右

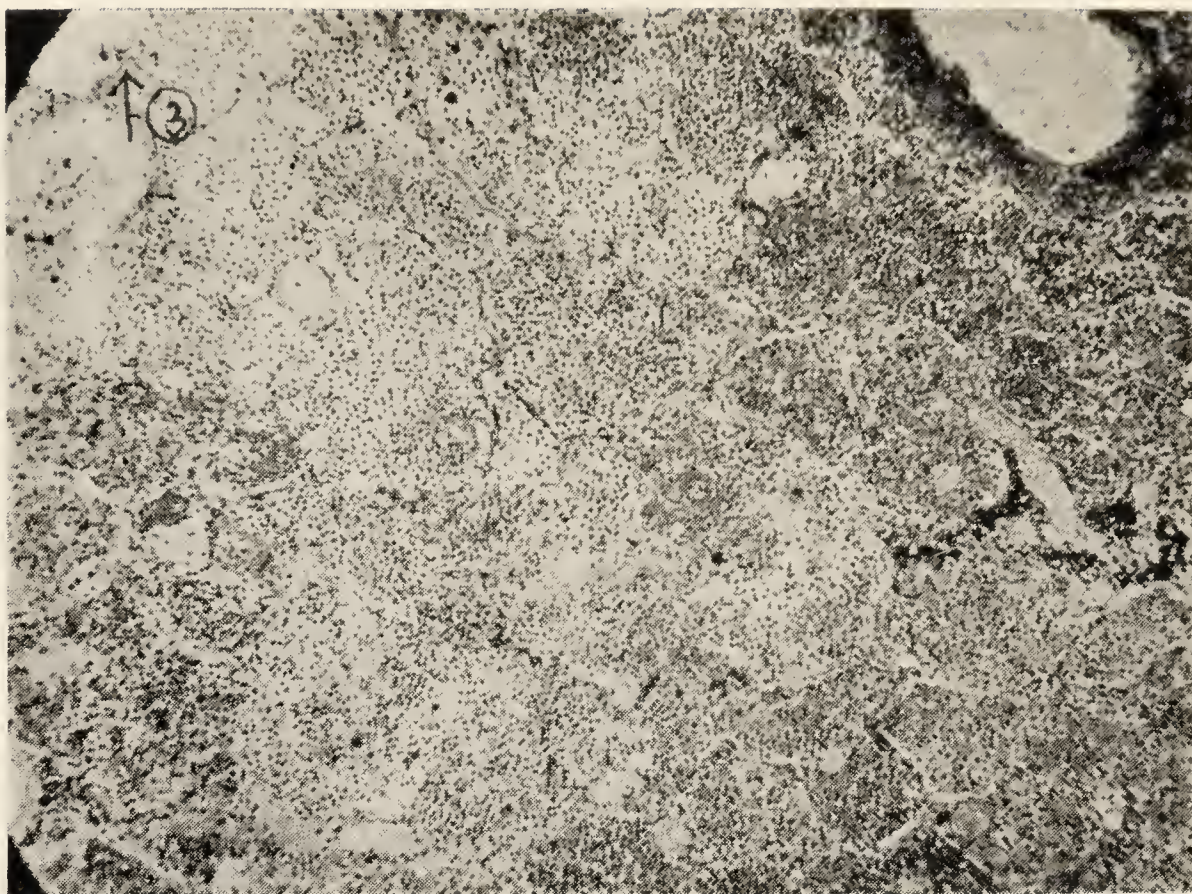


Section of Lung (human), Plague Pneumonia, mod. magnified. Sero-cellular exudation with some haemorrhage. Cellular exudation is not specially marked. Alveolar walls swollen and thickened.

(類人)纖組肺

液滲胞細血出微帶液出滲胞細清血有疫肺  
厚且腫壁胞肺明著甚不

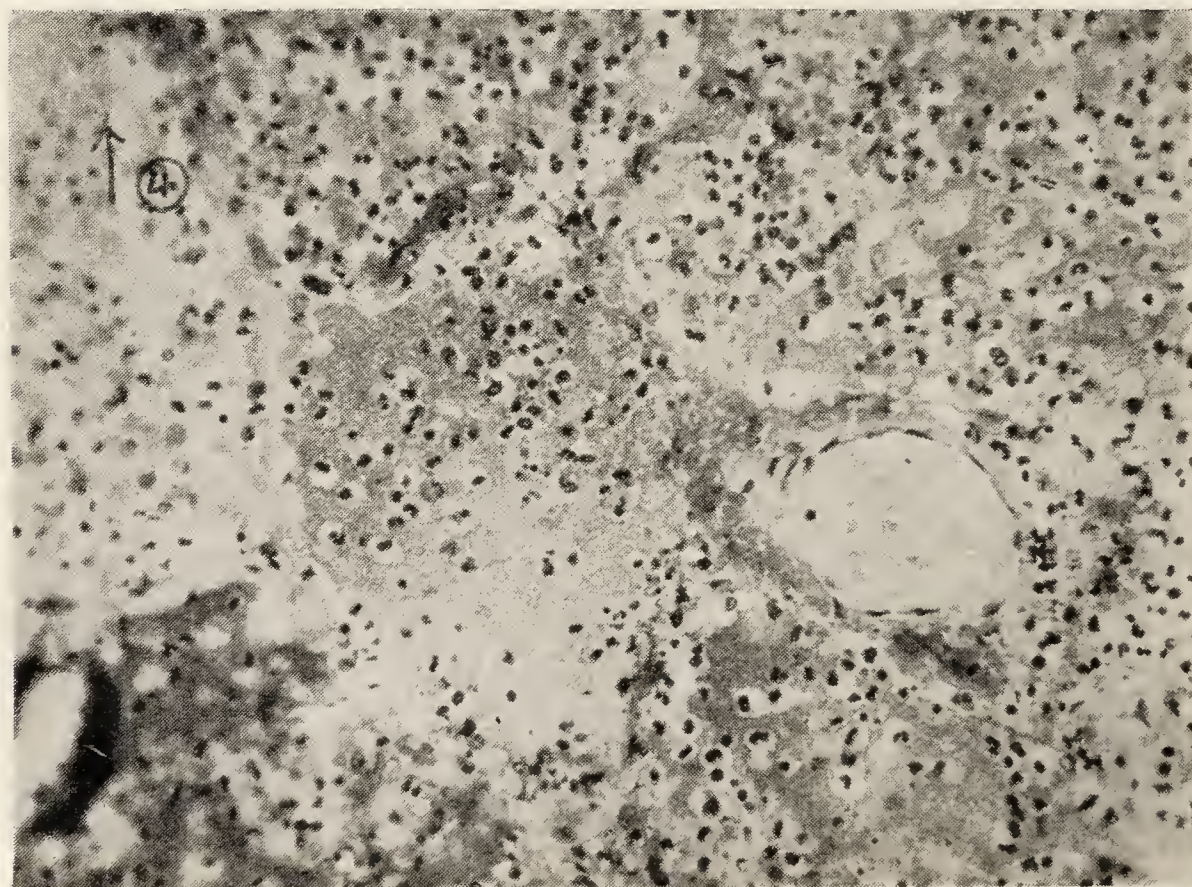




Section of Lung (human), Plague Pn., mod. magnified. Alveolar exudate rich in cells. Alveoli filled with B.P. Clearly marked black dots are cell nuclei. Cloudy effect due to presence of bacilli. Intensely black spots are carbon dust. The area surrounding blood vessel in upper right hand corner is marked by presence of both carbon dust and dense masses of B.P.

(類人) 組織肺

核胞細係點黑菌疫有內胞多甚內胞細在液出滲胞肺疫肺  
菌疫及埃炭有角右上繞圍管血有埃炭係黑著菌疫係濁混

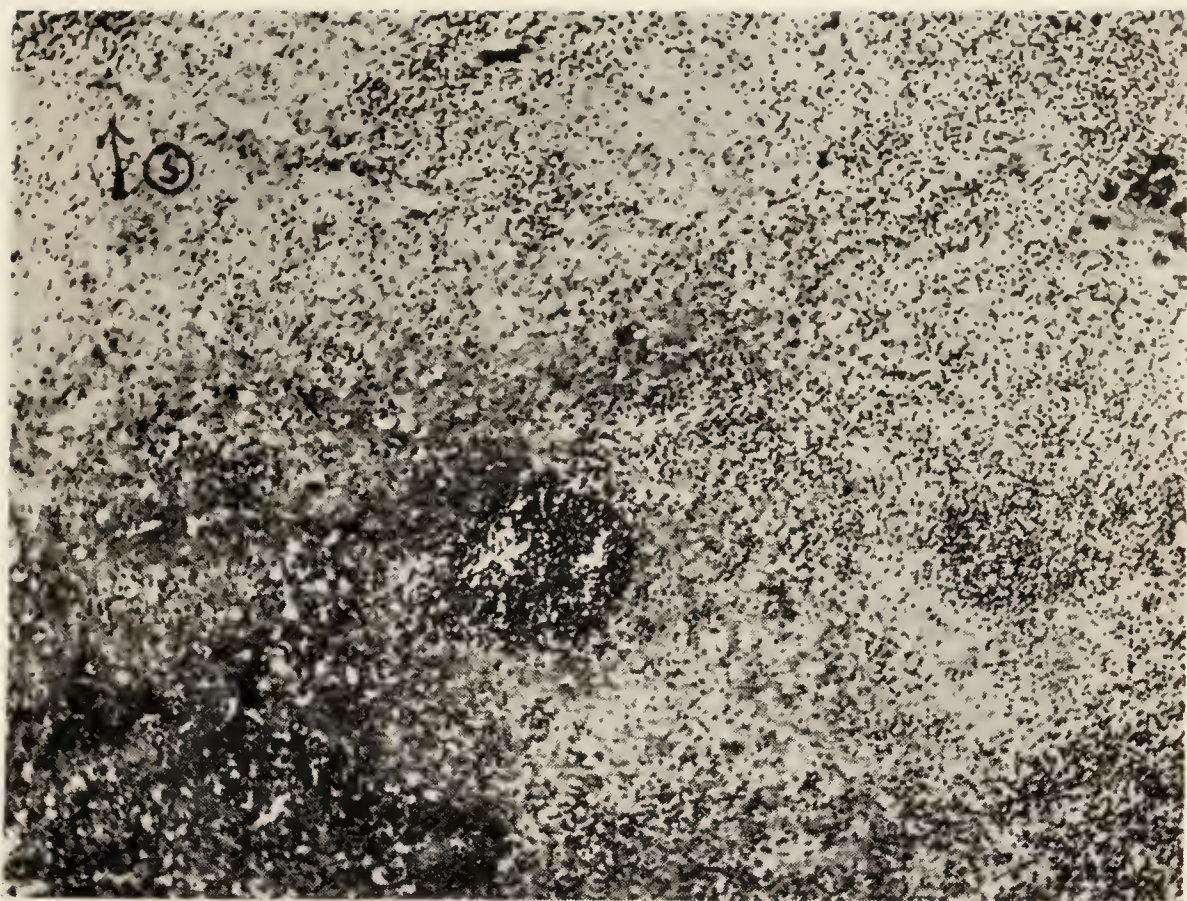


Section of Lung (human), Plague Pn., highly magnified. Alveoli filled with Plague bacilli and exudate containing many leucocytes. The small faint points are B.P.

(類人) 組織肺

菌疫係部濁暗小球血白含內液滲菌疫含內胞肺疫肺

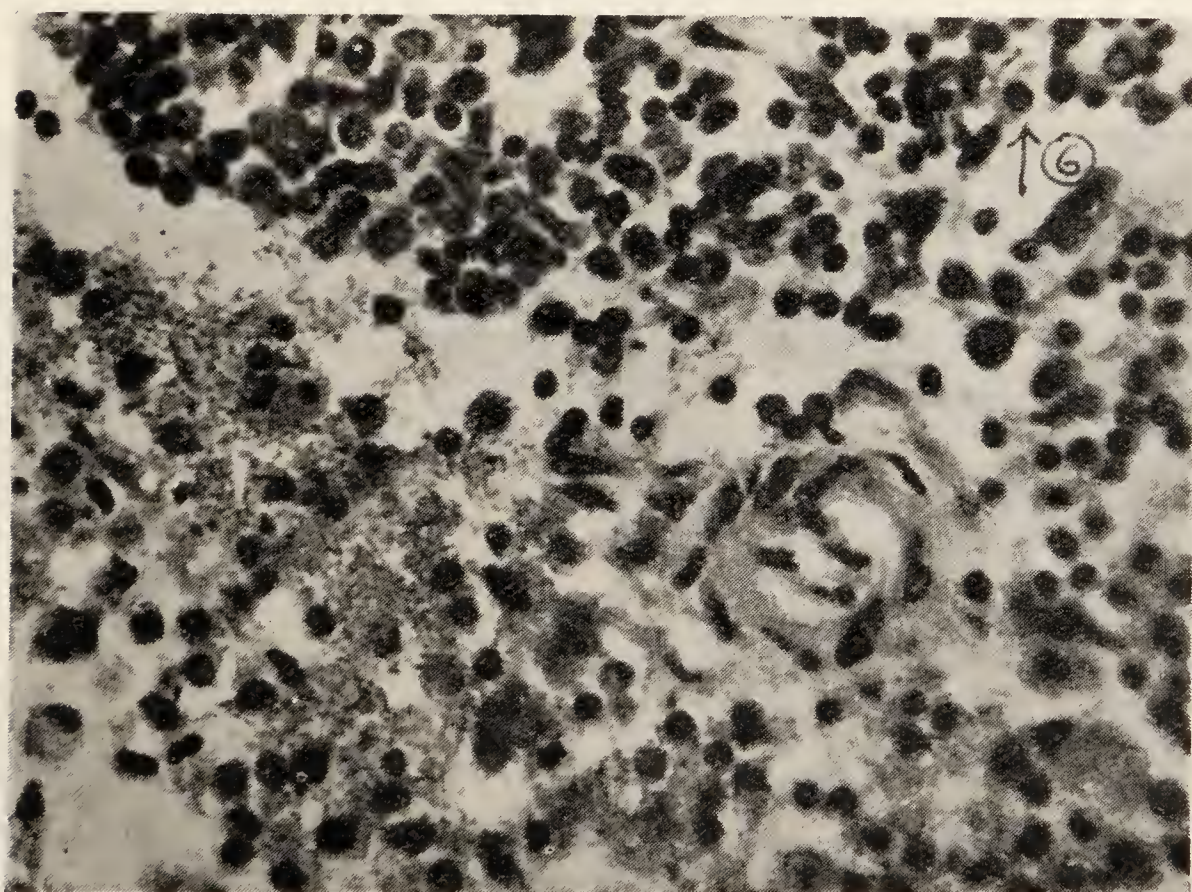




Section of Bronchial Lymph gland (human). Plague Pn., mod. magnified. Showing small lymph follicle. Cloudy effect due to B.P. Small black dots are cell nuclei. Larger black spots in upper right hand and lower left hand corners are carbon dust.

腺巴淋管氣

左下及右上在核胞細係點暗小菌疫係部濁混疫肺  
埃炭係點黑大

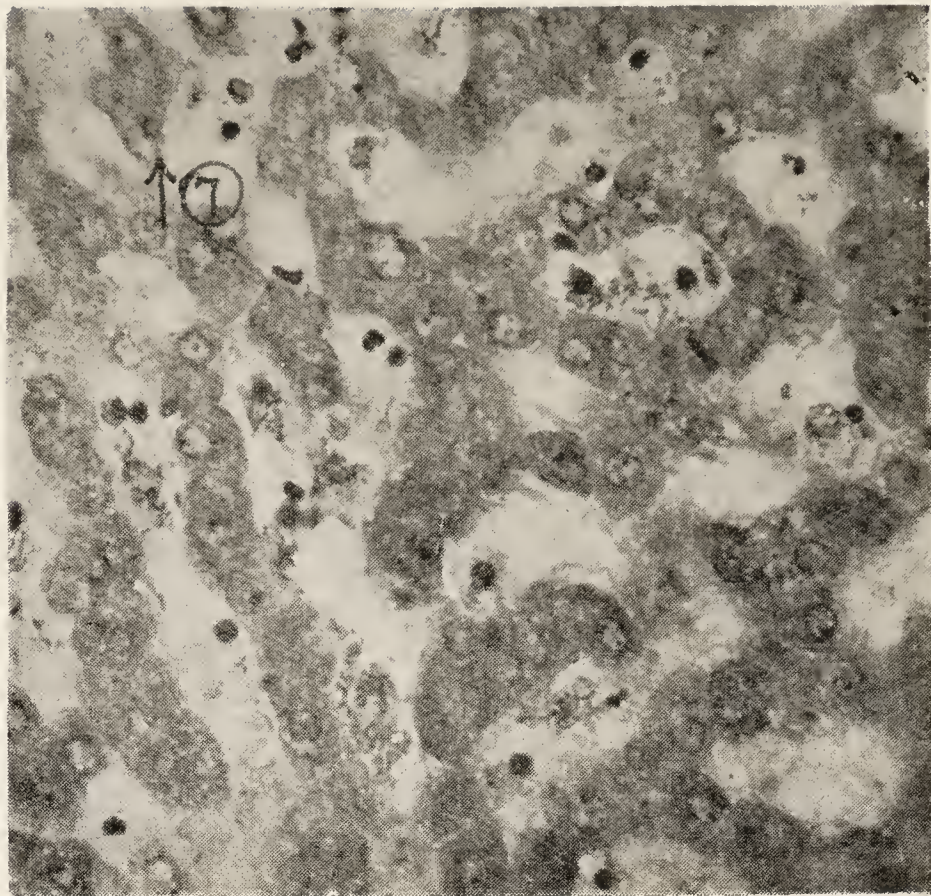


Section of Spleen (human) in Plague Pneumonia, highly magnified. In upper part, lymphoid cells of follicle. Note accumulation of B.P. in peripheral area of follicle. Towards right of centre is transverse section of small artery.

(類人) 纖組脾

部一之管血小係中右菌疫帶胞細巴淋部上疫肺





Section of Liver (human) in Plague Pneumonia, highly magnified. Blood capillaries contain numerous B.P. Inside some endothelial cells may be seen bacilli (phagocytosis).

(類人) 組織肝  
內胞細皮內在菌疫數無含內管血細毛疫肺  
(象菌喰即) 菌疫有見亦



Naturally plague infected tarabagan with all organs intact (Harbin Hospital Museum), found at Barun Zasulan, Transbaikalia, on July 25, 1923.

二十民於六十獺即 (存室物博院哈) 腑臟全其及獺旱疫鼠然天染  
得所地蘭蘇紫倫巴內加拜士蘭薦在五十二月七



occurs. The toxin of the virus may have caused vasomotor disturbance.

*Bacteria.* In nearly all organs plague bacilli are found in the blood vessels. Next to the lungs and lymph glands, these are most numerous in the spleen and liver. Gram-positive bacteria of mixed infection were noted to some extent in many cases, and sometimes were fairly numerous in the pneumonic areas of the lungs. These bacteria were sometimes observed also in other organs, especially in the lymph glands, spleen and even in the blood vessels of the liver, always, however, in relatively much smaller numbers than the plague bacilli.

*Phagocytes.* Phagocytes are found in the lumen of the alveoli, in the bronchioli and the bronchi of the pneumonic areas of the lung and also in the spleen. Could the lymph glands have been examined in the early stages of the infection numerous phagocytes would probably have been found there as well. The endothelial cells of the liver also act as phagocytes. Most of the phagocytes are mononuclear cells such as histiocytes and endothelial or reticulo-endothelial cells. The phagocytes within the lumen of the alveoli of the lungs have been generally regarded as desquamated epithelial cells, but the results of many recent examinations of other forms of pneumonia seem to indicate that these have originated from histiocytes or similar cells. To what extent the desquamated epithelial cells may act as phagocytes can only be determined by further study.

*Plague Bacilli in the Foetus.* Whether plague bacilli can pass over from the plague infected mother through the placenta to the foetus remains a question of interest. In the one case which has been examined no tissue changes characteristic of plague were found, nor did the stain reveal any plague bacilli in the tissue. It is stated by those who performed the post-mortems in 1921, however, that the cultivation examinations in this case showed positive results.

*Comparisons.* A comparison of these specimens with the pathologic-anatomic characteristics of the epidemic of 1911 is difficult to make, because pathological changes vary considerably in the same epidemic in different cases, and are also affected by several etiological conditions. A satisfactory comparison could be made after the examination of a great many cases. Because the foregoing examination covers only a limited number of specimens it does not permit of definite comparative conclusions. However, it may be safely said that the pathological changes in the two epidemics are very similar in nature, as is the mode of development of the disease.

NOTES ON THE HISTOLOGICAL LESIONS FOUND IN  
ORGANS OF NATURALLY PLAGUE  
INFECTED TARABAGANS.

(WITH 6 MICROPHOTOGRAPHS.)

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*(From the Laboratory of the Manchurian Plague Prevention  
Service, Harbin.)*

The only description extant of the morbid histology of plague in a naturally infected tarabagan is by Barikin, in *R. Vrach*, 1909, pp. 538-40. This Russian physician shot in the autumn of 1907 an apparently healthy tarabagan in the district between Lake Dalai Nor and Manchouli, which had been reported by resident Mongols to be inhabited by plague stricken animals and where recently a young Russian girl had died of bubonic plague with swellings in the left groin. Barikin dissected the animal and found the following gross lesions:—

“*Heart*, muscles ruptured and haemorrhagic;  
*Liver*, swollen and congested;  
*Spleen*, swollen and congested with small greyish nodes  
on section.

Both smears and cultures from the spleen showed plague-like bacilli. He also made sections of several organs and recorded the following histological features:—

*Heart*, haemorrhages between musculature and some rupture of muscle fibres.

*Liver*, no marked leucocytosis.

*Spleen*, parenchyma filled with pest bacilli, groups of which were surrounded by leucocytes. Some bacilli found in leucocytes. Necrosis not marked.

Other organs showed little visible change.”

A complete list of eighteen naturally plague infected tarabagans has been given in Table V of the article on Wild Rodents. Out of this number we were able to procure for microscopic examination organs from seven animals. These are (numbers refer to Table V):—



No. of Tarab.	Date found	Locality	Macroscopic lesions
T 5	Sept. 16, 1921	Kinkija (near Sektui)	{ Cerv. and Inguinal buboes. Haem. lungs, spleen enlarged.
T 11	June 8, 1923	Barun Zasulan	{ No buboes. Haem. foci in lungs. Spleen soft with nodes.
T 12	June 9, 1923	do	{ Cerv. buboes with abscess in left. Haem. foci in lungs. Saliv. gland. oedem. spleen and liver soft and enlarged.
T 13	June 23, 1923	do	{ Decomposed. Left Cerv. and Ax. buboes. Pneum. areas in lungs.
T 16	July 26, 1923	do	{ Cerv. buboes. Pneum. areas in lungs.
T 17	Aug. 24, 1923	do	{ Cerv. buboes. Many pneum. areas in lungs. Liver, spleen and kidney en- larged.
T 18	Sept. 1, 1923	Dauria	{ Cerv. buboes. Nodes in lungs Liver enormous with severa nodes.

The number of organs from which sections were cut for examination are not uniform, as the following table shows:—

	T5	T11	T12	T13	T16	T17	T18	Total.
Lung (a) .....	x	x	x	x	x	x	x	7
Lung (b) ....	x	—	—	—	x	x	x	4
Heart .....	x	—	—	—	—	—	—	1
Bubo .....	x	—	x (cerv)	—	—	x	x	4
Saliv. ....	x	—	x	—	—	—	—	2
Kidney .....	—	x	—	—	—	—	x	2
Spleen .....	x	x	x	x	x	x	—	6
Liver .....	—	—	x	—	x	—	x	3

The stains used were haematin and eosin (sometimes van Gieson) for general purposes, and carbol thionin (sometimes giemsa) for studying organisms. As alcohol had been used for preserving the specimens, the plague bacilli were easily distinguished by thionin. Unfortunately some of the specimens were not fresh when picked up, making a detailed study somewhat difficult, but the distinctive features did not vary very much.

*Lungs.* One remarkable feature is the presence of acute catarrhal-pneumonia (or conditions leading to it) in at least four out of our seven specimens. Here are seen the bronchi with infiltrated and congested walls, the lumen filled with mucus in which are crowds of plague bacilli; the air vesicles with swollen walls and containing catarrhal cells, leucocytes.

and fibrin, the interlobular septum presenting a hyaline appearance, and in places even haemorrhages besides intense congestion of the blood vessels. Red hepatisation is seen in two specimens. Some parts of the lung are quite solid, so much so, that the individual alveoli may not be distinguished from one another. Sometimes, the invasion of the bacilli in large groups has been so intense that the alveoli are compressed and even collapsed. Under high magnification these masses of plague organisms seem to form solid steams branching into and penetrating the lung tissues in all directions. Now and then red blood corpuscles and leucocytes are intermixed with them but not seldom the bacilli occupy more space than the blood cells. Where reaction is greatest, the capillaries are intensely dilated. Even the large blood vessels sometimes show great numbers of organisms inside them, interspersed here and there with coagulated fibrin.

The *pleura* in two out of seven cases shows thickening. In one of these, there is intense pleuritis, the lining being yellowish in colour and actually displaying haemorrhagic spots beneath with masses of plague bacilli. There is great effusion of blood in the alveoli near the pleural surface. Where the reaction is not so great, the fibro-elastic tissue is only thickened, but the underlying lymph spaces and small blood vessels are much congested.

In the *bronchi* connected with the pneumonic changes, the lining cells are swollen, disintegrated and often detached. As stated above, two specimens show considerable exudation, like the human cases, with muco-purulent matter present in the lumen, in which plague bacilli, red corpuscles and broken-down white cells are mixed together. Outside the epithelial lining, the bacillus pestis is most evident in the loose lymph space adjoining the muscular layer. In a few bronchioles masses of bacilli may be seen crowding into the subjoining lymphoid spaces.

Wherever the reaction is marked, the blood vessels are congested and show large numbers of red and white cells. At places the perivascular spaces are enormously dilated and filled with a network of fibrin enclosing numbers of plague bacilli. The cells lining the capillary walls are swollen and show proliferation.

In the specimens where no pneumonic changes are present, the usual signs of bacteraemia are found, with acute changes in the lung tissues by the side of congested blood and lymph vessels. The plague bacilli are more or less evenly distributed throughout.



*Lymphatic Gland.* Out of the total eighteen authentic cases recorded of naturally plague infected tarabagans, eight (perhaps nine) showed distinct swellings of the cervical glands. Three others being only remnants of animals, no examination was possible, while in the remaining six the condition of the cervical glands was not mentioned. Hence morbid changes may be expected in the specimens of buboes examined. The capsule of the gland is in every case more or less thickened with congested neighbouring blood vessels. Plague bacilli are present in varying numbers. The trabeculae are much swollen, the cells being flattened and enlarged. The cortical sinuses are somewhat enlarged, but not so markedly as those of the medulla, where numerous engorged blood vessels may be seen full of blood corpuscles and at places bacilli. Intense haemorrhage is present throughout the lymphoid tissue, especially the Malphigian bodies. As in the primary buboes occurring in human infection, large clumps of bacillus pestis, more deeply stained than surrounding tissues, are scattered all over the interior of the glands. One specimen (T 18) particularly shows solid masses of bacilli among numerous swollen blood vessels and haemorrhages beneath the capsule of gland. Apart from these clumps, the bacilli are present everywhere in large numbers, some mixed with the red cells in the capillaries. In several aspects, we witness a condition very similar to the changes encountered in true bubonic plague.

*Liver.* Three specimens were examined. One of these presented a truly extraordinary appearance. The capsule is not thicker than usual, but immediately beneath is much venous congestion. Under the low power, masses of deeply stained bacilli are dotted all over the section, invading and destroying the liver tissue and forming areas similar in appearance to metastatic growths in tumours. These are of various sizes and seem to have no immediate relation to the location of the lobules. Under higher magnification, the liver cells are seen to have withered away under the invasion, and present all the stages of granular degeneration, vacuolation and disintegration. Particularly is this change marked in the central zone, where considerable haemorrhages also take place. The central lobular veins are dilated and thickened and contain coagulated fibrin. The capillaries are everywhere dilated and filled with red corpuscles. The peripheral zone, though affected, still retains its individual appearance and is better stained. In other words, the condition of acute red atrophy is simulated. Leucocytes accumulate in vascular channels, while the endothelial cells present a granular appearance and are badly stained. The intervening connective tissue is oedematous. Under high power, plague bacilli are seen not only comprising

the big masses but are scattered in considerable numbers throughout the liver substance, principally along the capillaries. Where the big groups of bacilli have invaded the liver substance extensively, few normal cells are to be seen; only a reticulum of fibrous tissue is left.

The two other specimens show less marked changes, but the condition of acute plague septicemia (bacteraemia) is evident.

*Spleen.* Signs of acute congestion are present in all specimens examined. The capsule is in almost every case thickened, and minute haemorrhages occur in some places. The Malphigian bodies are swollen and not so deeply stained. Sinuses, both arterial and venous, are enlarged, and often filled with red corpuscles. The lymphocytes are unusually numerous, and enormous numbers of plague bacilli are scattered throughout the substance. The reticulum of adenoid tissue is swollen, the cells being flattened and stained indistinctly. No such big clumps of bacilli are visible as we have noted in the liver.

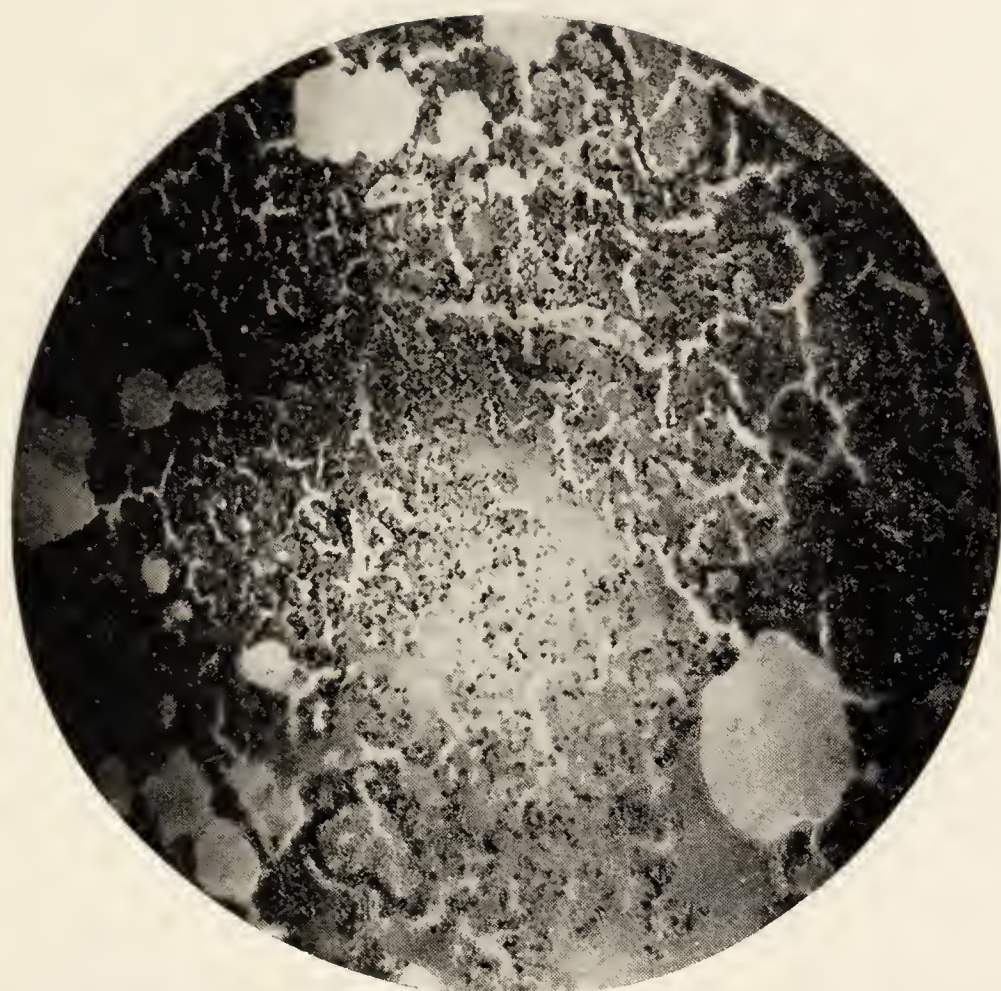
*Kidney.* Both specimens examined show considerable inflammatory changes. The capsule is either swollen or broken in parts. Small haemorrhages invade the cortex and medulla. The glomeruli are congested and faintly stained. Marked cloudy swelling is present in the cells of the tubules, vacuolation and granular degeneration being prominent in some parts. A few cells are seen dropping off from the epithelial lining. The walls of the arterioles are thick and hyaline. The cells lining Bowman's capsule show indistinct nuclei. Some golden brown pigment, mostly granular, is seen in many of the tubules. Plague bacilli are distinguished scattered along the blood vessels, but are not so prominent as in the spleen or liver.

*Heart.* Only one specimen (T 5) was examined. The bundles of muscular fibres are swollen, the striations being indistinct and presenting a cloudy appearance. Between these bundles the connective tissue is swollen and harbours many plague bacilli. The capillaries are enlarged and filled with red cells. Minute haemorrhages are scattered here and there. Apparently the toxic invasion looks nearly as severe in the heart as in other organs.

#### GENERAL REMARKS.

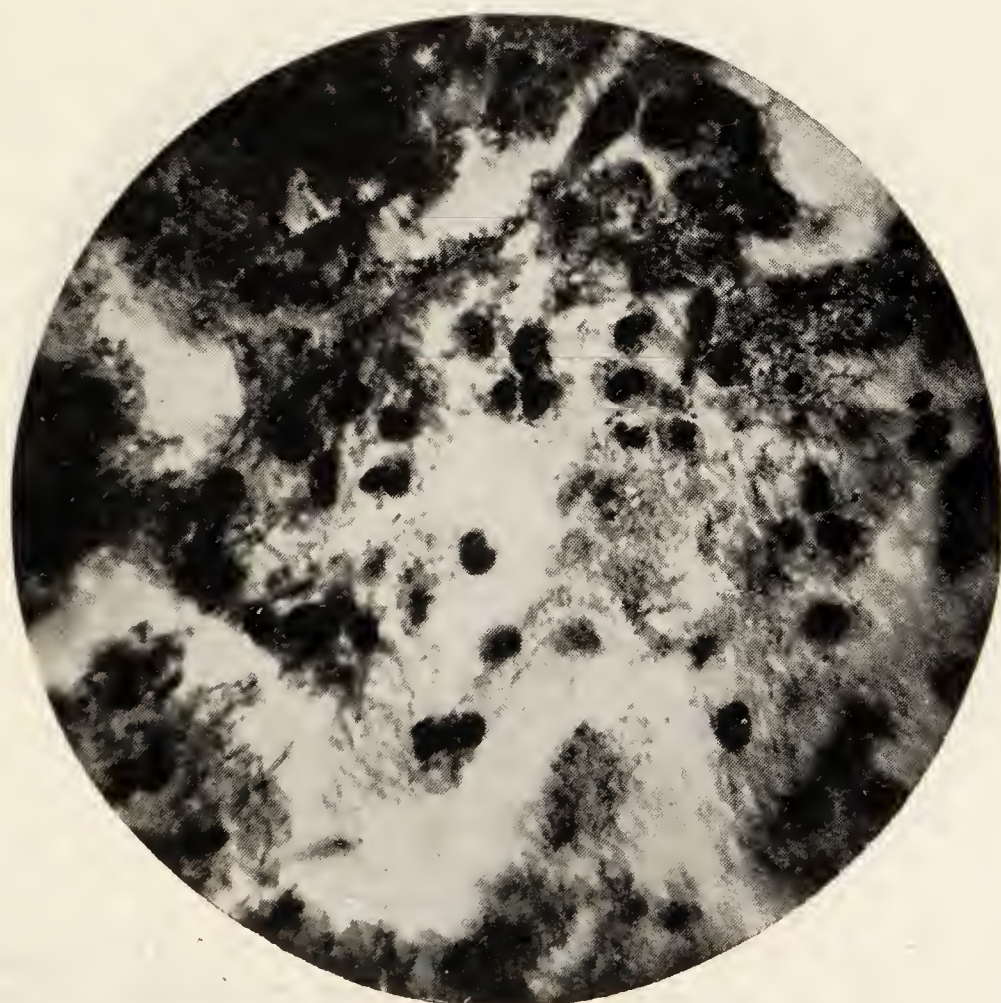
As referred to briefly in the article on Wild Rodents, we found, as far as macroscopical appearance was concerned, the lungs involved in ten out of eighteen cases, free in two, and unascertainable in the other six. It was with a view to find-





Microphoto of section of Lung (T 16) magnified 60 diam. Practically solid mass of pneumonia, with clumps of plague bacilli in midst. Many alveoli collapsed, and haemorrhages present.

肺 (六十 獺) 倍 大 廓 示 肺 炎 硬 變 中 央 富 疫  
菌 肺 胞 剝 爛 現 出 血 狀



Portion of above magnified 800 diam. Large numbers of plague bacilli are seen distributed among infiltrated lung tissue.

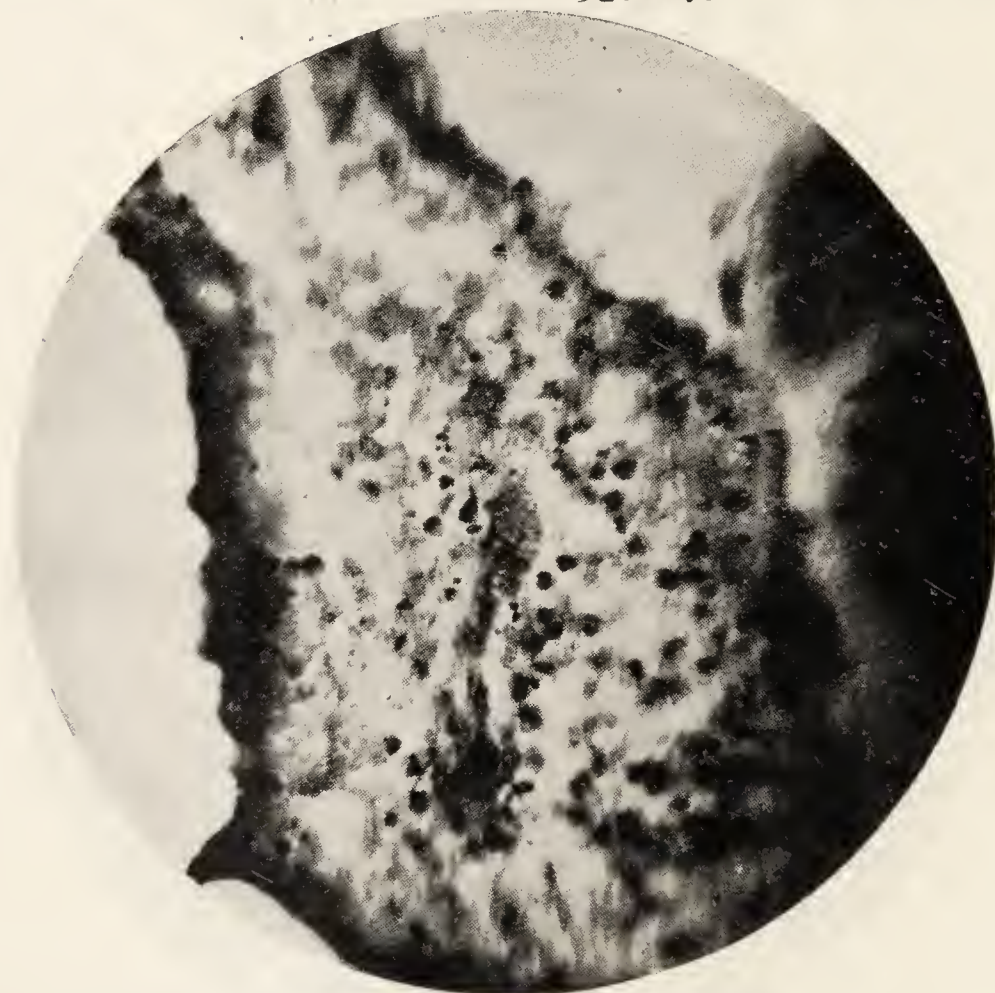
上 圖 一 部 八 百 倍 大 廓 示 肺 組 織 浸 潤 有 多 數 疫 菌





Microphoto of section of cervical bud (T 18) magn. 60 diam. Irregular groups of plague bacilli are seen in centre of field. Lymph sinuses engorged and haemorrhages everywhere.

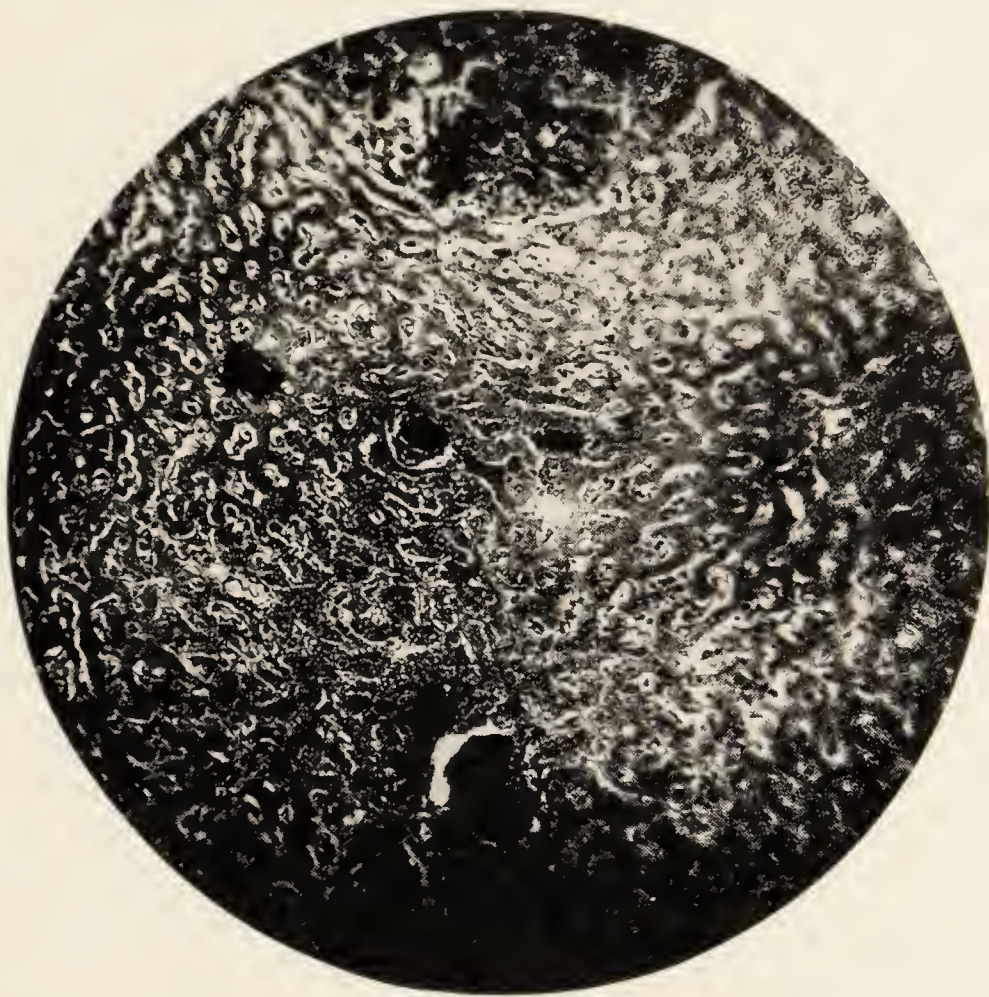
管巴淋菌疫類各部央中示大廓倍十六(八十獺)腫腺頸  
血出及血充現處各



Microphoto of section of a bronchiole in Lung of T 18, magnified 160 diameters. Lumen on left showing broken-down epithelium, beneath which are masses of plague bacilli. More bacilli groups adjoin muscular layer. (right of field.)

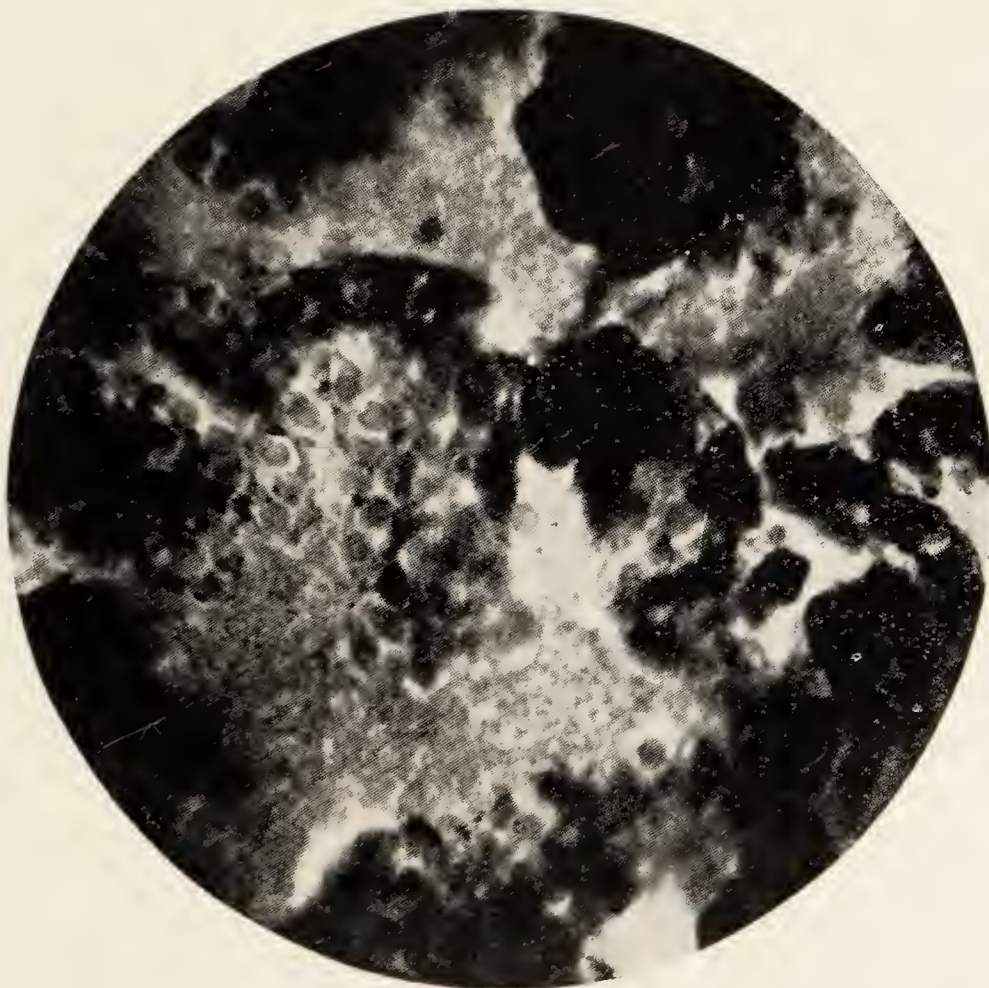
脫剝胞細皮上孔管示左大廓倍十六百枝管氣內肺八十獺  
(右本標)盛尤菌疫處連層筋與羣菌疫有下其





Microphoto of section of Liver (T18), magnified 60 diam. Central zone of lobule much disintegrated. At three corners of field, the characteristic nodules, consisting mostly of deeply stained plague bacilli are seen.

肝十六倍大葉中之部甚崩解於三個角部結節內含濃染疫菌



Same as above, magnified 800 diam. showing contents of a nodule. Enormous numbers of plague bacilli piled in centre.

同八百倍大節中央排列疫菌甚密





ing how far such lesions were primary and how far secondary that the present investigation was made. Unfortunately, we could not always be sure of the age of our specimens, as the carcasses were as a rule picked up in the fields without our knowing how long they had lain there. Still, as we had been waiting over ten years to obtain such specimens, we had to be satisfied with the materials at our disposal.

In every one of the seven animals, whose organs were examined by us histologically, a condition of general plague septicemia or rather bacteraemia was present, showing acute degenerative changes in the tissues and corresponding to those met with in ordinary acute bubonic infection. The microscopic lesions noted everywhere followed closely those we are used to in cases among rats, and so need not be repeated. But the remarkable features observed in the respiratory tract seem to point to something deeper than what is so commonly encountered in rat plague. The suspected appearance of pneumonia, when viewed with the naked eye, was fully confirmed under the microscope. In four at least of the seven cases examined, a condition of acute broncho-pneumonia exists, giving the general characteristics of this disease. The acutely inflamed bronchus with its swollen epithelial cells, the distended blood vessels and even haemorrhages with the ever present crowds of plague bacilli permeating almost everything, and finally the mucopurulent secretion in the large and small bronchioles where red corpuscles and plague bacilli are found together reminds one vividly of similar changes observed in pulmonary plague. Perhaps this feature is present in only 40 per cent. of the cases, but even this number is sufficient to bring up the query, "How do the tarabagans infect one another in nature?" Through insects as in rat plague or directly as in cases of primary lung infection? The lesions so far observed seem to point to the former, the lung conditions being due to secondary invasions from other quarters.

But the close association of tarabagan plague with primary pneumonic outbreaks in man, as distinct from rat plague which generally leads to bubonic cases in man, should always be borne in mind. In this respect, the observations made in California upon the pest among squirrels where a tendency to pneumonic invasions of the lungs also exists, offer an interesting parallel.

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## THE ORIGINAL HOME OF PLAGUE.

(WITH ONE SKETCH MAP.)

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## II. HISTORICAL EVIDENCE.

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In discussing this problem, two roads could be chosen to reach our goal. One method is the *historical*, by which the history of plague from the earliest times may be studied and traced to one or more common sources. The other method is the *geographical*, by which certain localities known as endemic foci may be collected and criticised so as to ascertain how far they are really independent of one another.

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## 1. GEOGRAPHICAL EVIDENCE.

The geographical evidence is first taken. We start with the endemic centres not as they are known to us nowadays, but, wherever possible, as they were mentioned in the pandemic starting in 1894. This is done because new localities have been turned into "endemic-foci" since then by the



introduction of extraneous infection, e.g. South Africa, California, Java, Senegal, etc. In other words, the distinction between primary, autochthonous or indigenous plague foci and secondary plague foci has to be borne in mind. That this is not always easy, the following pages will show, but it is the most satisfactory for the purposes in view.

A good beginning may be made with the primary endemic foci known about the beginning of the present century. These are:—

A. *In Africa.*

1. Benghazi (Tripolitania)
2. Central Africa (near the Victoria Nyanza).

B. *In Asia.*

3. Assyria (Arabia).
4. Western Asia with center in Kurdistan.
5. Kumaon and Gurwhal (N. W. India).
6. Part of Yunnan Province (China).
7. Transbaikalia and Outer Mongolia.
8. Inner Mongolia.
9. Thibet.
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11. Persia.
12. Astrakhan and adjoining territories.

A. ENDEMIC PLAGUE FOCI IN AFRICA.

1. Benghazi is the ancient Cyrenaica in the province of Tripolitania, North Africa. Plague was epidemic in Tripoli from 1856-1859 and again in 1874; no definite origin of these outbreaks was traced<sup>(1)</sup>. There were not only epidemics in the 19th century up to 1843<sup>(2)</sup>, but the district seems to have been already affected at the time of the Black Death, when the disease spread from Egypt in a westerly direction along the African Coast. Previous to this already there was an outbreak in 1270 in the adjacent Tunis among the army of Louis XI. To return to the 19th century, there was another outbreak in Benghazi in 1892. A Medical Commission was sent out from Malta to investigate the disease. It is true that this commission considered the outbreak as one of "spotted fever," but as the cases had "boils and even axillary abscesses," the affection was suspected to be plague. Moreover the appearance of the outbreak coincided with the arrival of a number of Bedouins from the interior. These people—about 20,000—were driven from their homes through famine. It is

(1) Local Gov. Board Rep. 1879/98, pp. 248-249.

(2) Simpson, A Treatise on Plague, p. 38.

true, that a preexisting infection might have gained new impetus among these poverty stricken people, who were crowded together, but no evidence was forthcoming in this respect. "As the disease is also endemic in Central Africa, it could obviously travel under circumstances northwards as far as Tripoli."<sup>(3)</sup> This opinion was endorsed by Koch<sup>(4)</sup> and by Bourges.<sup>(5)</sup>

No further outbreaks were recorded in Tripolitania until 1913 when some cases were observed at Benghazi and in the villages adjoining it<sup>(6)</sup>. Its plague character was officially denied, but nevertheless there seems to have been no doubt about it. The disease appeared exclusively among the Soudanese blacks. No definite source of the outbreak was given, but it appeared that Benghazi formed the starting point for overland caravans to Egypt. Testi<sup>(7)</sup> mentioned an outbreak of plague at Benghazi in the second half of 1913 which was not completely suppressed until the middle of 1915. He thinks the disease imported from the interior and kept up by continued fresh importations from there, stating that the local conditions are against the spread of the disease to any great extent by rats. In 1913 plague was observed at other places of Tripolitania as well, e.g., Derna and Tripoli. The disease appeared again in 1914 at different places, in 1916 in Tripoli among the Italian troops, in 1917 at and near Benghazi, Mazzone<sup>(8)</sup> who described the 1917 outbreak, stated that there was no previous epizootic, and infection seems to have been introduced by human agency.

## 2. *Central Africa.*

In 1897 Koch and Zupitza discovered an endemic focus in the then German East Africa, between the Kagera-Nile and the Victoria Nyanza.<sup>(9)</sup> Zupitza<sup>(10)</sup> visiting the spots observed that plague existed not only among the wild natives but among rats infesting the dark banana growths. This plague focus is apparently of very old age. A long series of outbreaks up to the present year can be traced back to this endemic centre. While stating this we do not want to convey

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(3) Local Gov. Board Rep., l. c. It is important to note that also the 1858 and 1874 outbreaks started among hunger stricken and badly housed nomadic Bedouin tribes.

(4) D. Med. Woch. XXIV, 1898, No. 28.

(5) La Peste, 1899.

(6) Local Gov. Board Rep. 1914/17, pp. 115-116.

(7) Giorn. Med. Milit., 1916, No. 10, pp. 737-771.

(8) Gaz. Osped. et d. Clin. 1919, April, p. 254.

(9) D. Mediz. Wochenschr. No. 28, 1898.

(10) Zeitschr. f. Hyg. und Inf. Kr. XXXII/2.



the impression that they originated at Kisiba, because it seems that plague is widely endemic in the Eastern parts of Central Africa.<sup>(11)</sup> As is quite usual when discussing the history of plague, the German authorities claimed that the infection came from the English territory and vice-versa. "The truth probably is that the endemic area of plague on the Western side of Victoria Nyanza included a portion of the German as well as the British territory, plague being endemic in both."<sup>(12)</sup> Furthermore it must be stated that while we owe to Koch and his collaborator the scientific establishment of the diagnosis, mention of the ravages of plague had been made by previous observers, e.g., by Lugard in 1893.<sup>(13)</sup> It would lead us too far to enter into a detailed discussion of this problem which is further complicated by the circumstance that for some of the outbreaks east of the Victoria-Nyanza importation from India is probable or possible. For our purpose suffice it to state that there exists in East Africa a large endemic area of old standing. It is not impossible that the focus in Benghazi is fostered from it, especially in view of the evidence collected by different observers at different times. Regarding the plague focus in Central Africa it is at present difficult to explain its origin. Notwithstanding contrary statements (Sticker) there is no doubt that general considerations would lead us to look for the original home somewhere in Asia, which is the first habitation of rodents. It would of course be possible for plague to reach these regions via Egypt, this apparently ancient homestead of the pest. The most probable explanation will perhaps be found by reviewing the two historical pandemics, when the spreading power of the disease was noticed at its height. There is much evidence that during the Black Death Upper Egypt was attacked. But one should be wary at jumping to this conclusion, as the Sudan, lying between Upper Egypt and East Africa, seems to have been free of the pest from the earliest times up to 1911.<sup>(14)</sup> On the other hand, the earliest authentic pandemic recorded, namely Justinian's plague, was stated by Evagrius to have started in Aethiopia, a locality not distant from Central Africa. Payne<sup>(15)</sup> who considers Egypt one of the original homes of plague, states that "possibly, if we could follow the history far enough back, we might find, that the African was a colony of the Asiatic plague." There are many still doubtful points in

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(11) Hodges Trop. Dis. Bull. I. pp. 289-91.

(12) Loc. Gov. Board Rep. 02/03, p. 312.

(13) Ibid. 1879-1893, pp. 249-251.

(14) Balfour, Sandwith, Lancet Nov. 1911, p. 1262.

(15) St. Thomas's Hosp. Rep. Vol. XVII, p. 8.

the history and geography of the pest in Africa, which may well deserve comprehensive study. Thus not only for Tripolitania, but for other regions in north and northwest Africa, an importation from the interior seems possible or even probable. This seems true in the case of Morocco, where the outbreaks in its northern part appear to be connected with the Draout tribe who live in the South-west of that country but migrate regularly northwards.<sup>(16, 17)</sup> It might be interesting to search for some connection between these supposed plague districts and the endemic areas in East Africa.

It is difficult to trace the mystery of African plague without some mention of the affection in the island of Réunion and Mauritius. An excellent survey of this problem is given in Bruce Low's report for 1898-1901<sup>(18)</sup>. A disease called by the name "Lymphangite infecteuse" had existed at Réunion since 1864 or even earlier and was supposed to have been imported in 1864 from Bombay. When Thiroux announced true bubonic plague on the island in 1894, it was at once asked if the case occurring since 1864 were not that disease also. This same suspicion was raised in the case of Mauritius, where during 1866-1867 the "lymphangite infecteuse" also raged in epidemic form and killed 40,000 persons.

*Egypt*: Though visited long ago by plague which spread to other countries (the first authenticated pandemic, Justinian's plague, is said to have started from Pelusium) Egypt does not seem to be a real endemic centre. Simpson<sup>(19)</sup> points out, that when Egypt was cut off politically and commercially from the East, no plague appeared there and emphasizes that after the introduction of quarantine measures in 1831 the pest soon disappeared. This view is also maintained by other authors, e.g., Kolle<sup>(20)</sup>. Thus it seems that plague appeared in Egypt only when introduced from outside, lingering often for considerable lengths of time and conveying the impression of endemicity. The disease was probably introduced from the East. Nevertheless it is not easy to trace definitely even the 1899 outbreak—whether from India or the Far East or the nearer shores of Arabia. Anyhow no evidence is procurable to show that any of the Egyptian outbreaks arose from the endemic centre in East-Africa.

(16) Sacquépée and Garcin Arch. de Med. et Pharm. Milit. 1913, pp. 561-579.

(17) Remlinger, Paris Medic., 1914, pp. 234-235.

(18) Loc. Gov. Board Rep. 1898-1901, pp. 181-196.

(19) l. c. .... pp. 176-177.

(20) Wilson's Infect. Dis., p. 760-761.



## B. ENDEMIC FOCI IN ASIA.

3. *Assyr*, Western Arabia, especially its high plateau, is generally considered as an endemic focus. Epidemics have been known in this region since 1815<sup>(21)</sup>, but as Kremer<sup>(22)</sup> points out, an outbreak was already recorded there in 1157. This was probably not a solitary or indigenous one, as plague was very active and widespread at that time. It seems that the invasion was not restricted to Assyr alone, but travelled along the coast and was present in Jambo, Jeddah and up to Mecca<sup>(23)</sup>.

The endemic focus in Assyr is important because caravan routes to Mecca pass through it, as also others from Southern and Western Arabia.

Nothing is on record of epizootics prevailing among rats and other rodents in this area<sup>(24)</sup> but it must be added that the evidence is also scanty for human outbreaks.

No records of Assyr were obtainable after 1906, although outbreaks were still reported at Jeddah, etc.

4. *Western Asia*. This "large area, comprising Persian Kurdistan and adjacent parts of Persia, Turkish Kurdistan, and parts of Irak or Mesopotamia on the banks of the Tigris and Euphrates, including Baghdad"<sup>(25)</sup> can be considered as a whole. Tholozan<sup>(26)</sup> had already pointed out, that the real endemic centre of this region was to be found in the mountains of Kurdistan. This statement has not been contradicted.

This endemic centre is doubtless one of the most important ones. It has been active up to this day and the oldest records of plague can be traced back to it. If one believes the outbreak mentioned in the Bible<sup>(27)</sup> to be plague, then it would seem that the affection, starting during the war between the Israelites and Philistines and apparently connected with the "mice of the field" had started from the endemic focus in Western Asia. This might also be true of the plague of Justinian (A.D. 542). Pelusium which had considerable commercial intercourse with these regions could then be looked upon as a distributing centre.

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(21) L. G. B. Rep 1879-98, p. 203.

(22) Ueber die grossen Seuchen des Orients, etc. 1880.

(23) Report of German Plague Commission, p. 351.

(24) Martin, Lancet, Nov. 1911, p. 1250.

(25) Payne in Albutt and Rolleston, A System of Medicine, 1910, p. 386

(26) Histoire de la peste bubonique en Perse, 1874.

(27) 1. Samuel ch. V-VI.

Martin<sup>(28)</sup> states that there are no records of epizootics prevailing in this endemic area. There seems however no doubt that rats abound in the rice fields of Mesopotamia<sup>(29)</sup>, and Avicenna (A.D. 908-1037) also recognised its connection with epizootics. During the World War (1914-18), outbreaks among the rats of Mesopotamia were noted.

It is known to students of plague history that former authors endeavoured to differentiate between two varieties of plague "Western Asiatic" and "Indo-Chinese." These authors maintain that the two sets of endemic centres are geographically independent of each other, and also that there are marked distinctions regarding their epidemiology and symptomatology. There is however really no difference between the two "strains" of bacilli, and it can be further shown that the two endemic foci, although far removed from each other, are epidemiologically connected. This can be demonstrated by first describing the endemic foci in the Far East and then turning to the connecting links.

5. *Kumaon and Gurwhal.* An account of plague in this region situated in the North-West of India on the slopes of the Himalayas, is given in the Loc. Gov. Board Report<sup>(30)</sup> and in the report of the German Plague Commission.<sup>(31)</sup> This focus received much attention at one time, when it was seriously considered as the original focus of the outbreak in Bombay (1896).

The first cases recorded were in 1823, but various observers (Hutcheson, Hirsch, Plank) maintained that the disease is of much older standing and "has existed in all probability in the hill tract of India and China from time immemorial."<sup>(32)</sup> Sticker is inclined to trace Indian plague remarked upon by Ibn Batuta (1325-1351) to this focus.

This locality is highly situated and sparsely populated, most of the inhabitants living 3000-6000 feet above the sea; they are poor and dwell promiscuously with their cattle. The physical character of the districts is such that the conditions for a spread of the disease are rather unfavourable; furthermore the population is well acquainted with the disease and flies in time of danger from the affected villages into the forests. The only thing which may seem likely from a theoretical point of view is the existence of a few places within the districts visited by pilgrims, but this is of little practical

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(28) l. c.

(29) Waum, quoted in Zabolotny, *Pestis bubonica* (1907).

(30) L. G. B. Rep. 1898/99, pp. 199 and foll.

(31) l.c., pp. 12-23.

(32) Hutcheson, Indian Medical Congress, Calcutta 1894.



importance. It is true that the 1823 outbreak was said to have started in the holy places (Kadainath or Hurdwar), but on the other hand observers have noted very few infections among visiting pilgrims. Besides, plague was rarely traced from there into the plains of India: only three outbreaks—(a) (1825-28)—in Housi (province Delhi), (b) (1836-38) in Bareilly (province Rohilcand) and (c) (1863) in the district Moradabad—have been known to start from this centre. Our knowledge about *a* is scanty, being based solely upon a statement by Skinner as quoted by Hirsch. The authenticity of *b* seem dubious<sup>(33)</sup>. There, thus, remains on record only *c*, which caused 8000 deaths.

Different theories were formed for the origin of plague in these districts. It is maintained that the disease “is imported from time to time from Thibet into Kumaon and Gurwhal and is not, strictly speaking, endemic in these districts.”<sup>(34)</sup> The German Plague Commission was rather sceptical about this theory, stating that there is nothing on which it can be based; and pointed to the fact that the region is separated by the Himalayas from Thibet, whose passes are traversable for four months in summer only. Yet the possibility of Thibet being a focus has to be seriously considered. The German Plague Commission seemed rather inclined to trace the cause of the endemicity to the wild rodents in Kumaon and Gurwhal (*Arctomys*, *Leggada Jerdoni*). Regarding the domestic rodents Planck<sup>(35)</sup>, obtained (up to 1877) out of 40 affected villages only 8 positive findings of infected rats. He described the rats seen by himself as “a more delicate looking grey species.” Hutcheson comes to the conclusion that “spontaneous outbreaks are frequently associated with and sometimes preceded by a great mortality among rats, mice and other rodents.”

It is not quite settled where the present pandemic in India originated. Three possibilities may be considered:

- (a) Kumaon and Gurwhal.
- (b) By sea from the West (Persian Gulf—endemic center in Mesopotamia—or by returning pilgrims from Jeddah—endemic centre in Assyr).
- (c) By sea from Hongkong.

The last is the generally accepted one. Nathan points out “that there was no unusual prevalence of plague in Mesopotamia at the time of the outbreak in Bombay.” On the

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(33) Nathan, The Plague in India. Simla, 1898, vol. I, p. 85.

(34) Loc. Gov. Board Report 1879/98, p. 216.

(35) Rep. German Plague Comm. p. 16.

other hand plague prevailed at that time in Hongkong and sea traffic was not properly controlled. Certainly no quarantine measures were enforced in Bombay against Hongkong. A remark by Mueller<sup>(36)</sup> may be quoted, "Whether the plague of Bombay (1896), of Cutch and Kathiawar (1815-21) and of Pali, Marwar and Deogar (1836-38) originated in Kumaon and Gurwhal or in Hongkong is uncertain, but in any case the original focus whether by sea or land must be traced to the highlands of the Himalaya."

6. *Yunnan*. It would be rash to consider the whole of this mountainous province situated in the south-west corner of China as endemic for plague. For instance Vadon<sup>(37)</sup> states that he has not seen a single plague case in Yunnan-fu during four years and adds that Yunnan should no longer be regarded as a focus of endemic plague<sup>(38)</sup>. Vallet<sup>(39)</sup> also denies that the plague ever occurs there. This is confirmed also by a personal communication by Mr. Graham, a missionary, living in the province over 30 years. He wrote us that "there is less of plague now in the province than there used to be," and stated that he had not noted any plague in Yunnan-fu since the big outbreak of 1890; the Mengtsze plain during the first 15 years of his stay was visited practically every year by plague, but since then it had totally disappeared. In spite of these statements we believe that Yunnan plays an important part in the epidemiology of the disease, though not as an indigenous endemic area, but only as a connecting link or as distributing centre. Outbreaks have been mentioned in this province for a long time. Minakata<sup>(40)</sup> quotes as authority the Chinese explorer Hung Liang-Kih (1736-1809) who described plague among rats and human beings in that region. The old age of plague in these parts is confirmed by other observers. A picturesque, though not quite accurate account is given in Simpson's book<sup>(41)</sup>. Rocher<sup>(42)</sup> who visited the province in 1871 and afterwards, found that plague had been known there since 1840, but that *long before that time it had existed in the western part of the province without prevailing epidemically*. Rocher mentions the possibility of infection by caravans or pilgrims from Burmah. It seems doubtful if this theory can be upheld since we are unable to obtain records of early cases in Burmah. Yunnan is connected by caravan routes

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(36) Die Pest, Wien, 1900, p. 105.

(37) Ann. de Hyg. et de Méd. Colon., 1914, pp. 501-525.

(38) China Med. J., 1914, p. 398.

(39) Bull. Med. Franco-Chinois, 1921, pp. 55-62.

(40) Nature, Febr. 1899.

(41) l.c., pp. 48 and foll.

(42) Notes sur la peste au Yunnan.



with Thibet and it is more probable for plague to have a permanent home in sparsely populated mountain regions than in populated plains. It appears that Rocher himself had doubts about Yunnan being a primary plague focus, saying that the disease was brought there from the mountainous regions west of Yunnan, its central and eastern parts having been affected since the Mohammedan Revolution of 1853. The capital city is said to have been attacked for the first time in 1872-73.

7. *Transbaikalia and Outer Mongolia.* Only an outline of the plague in these areas will be given here, as the course of the disease in these parts is fairly well known through our researches.

The first mention of a disease among the tarabagans and spreading from them to human beings, was made by a layman, Tsherkasoff, in his book, "Memories of a Hunter in Eastern Siberia 1856-63."<sup>(43)</sup> He writes: "There are years, in which the natives do not eat the tarabagans, because the latter have an epidemic disease—they die like flies and many incautious natives, satiating themselves with infected tarabagans, not rarely pay with their lives." The first reports made by medical men in this respect are those of Bjeliavski and Rejshetnikoff.<sup>(44)</sup> Although these authors and many after them did not see sick tarabagans, they encountered human cases, but the bacteriological proof was wanting. The first bacteriological examinations in man were not made until 1905. It seems that the oldest human outbreak on record occurred in 1863 at Zagan-Oluevski (Transbaikalia), though other cases had evidently happened much earlier in those regions. Many authors consider that plague in Transbaikalia is of very old age. A good survey of this question for instance is given by Wassilewski.<sup>(45)</sup> He points out that the natives have known plague for generations, that they possess a working knowledge of the disease and adopt elaborate precautionary measures for fighting it. It is furthermore mentioned that the tarabagan, its habits and disease are part and parcel of the folk-lore of the Buriats and Mongols and that the disease among these animals and man is recorded in old Thibetan sacred books.

Among the numerous outbreaks, two small ones are on record where the disease started purely in the present territory of North Manchuria, namely 11 cases (1905) in the districts of the "Solons," one case (1923) near Jakoshih—both former Mongolian districts. In these instances the human outbreak was probably preceded by an epizootic among tarabagans, whose *bootans* (burrows) are plentiful around these parts.

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(43) German Edition Leipz. 1884.

(44) Vestnik Obst. Guig., 1895.

(45) Ibidem, 1915. pp. 178-201.

The endemic focus in Outer Mongolia comprises a very large area. This can be shown by two extreme instances. (1) An epidemic occurring in 1899—450 versts south-east of Urga;<sup>(46)</sup> (2) A smaller one recorded in 1899—200 versts east of Kobdo.<sup>(47)</sup> The distance separating these two points is about 1050 versts (700 English miles).

8. *Inner Mongolia.* Zabolotny<sup>(48)</sup> and other Russian observers believe that the district of Weichang (lat. 42° long. 118°), the famous Imperial hunting park of North China, is a true endemic centre of plague.

Dudchenko,<sup>(49)</sup> who has devoted much time to the study of the tarabagan problem in Siberia, considered that the hibernating habits of the animal helped to limit the spread of the disease, inasmuch as the sick one usually stayed outside their burrows to die. In trying to assign a reason for the almost yearly appearance of the epizootic among tarabagans, he laid stress upon the regular introduction of plague by the pilgrims passing through Weichang. It is true that Catholic missionaries reported cases of bubonic plague at Weichang as early as 1888, and Zabolotny on his visits there in 1898 bacteriologically confirmed the disease.<sup>(48)</sup> But since that time nothing has been heard, and Chinese medical officers stationed in the neighborhood have not reported any cases for nearly 25 years.

In regard to the 1917-18 (Shansi) epidemic a Russian observer declared the presence in August 1917 of a "winter sickness" which occurs periodically in the Ordos country and in Inner Mongolia.<sup>(50)</sup>

9. *Thibet.* Another starting point of the Shansi epidemic (1917) may be Thibet. Parry<sup>(51)</sup> states that the invasion started in a monastery called Mai-Uh and was carried afterwards to Taochow, Kansu, (China). Although he failed to reach the place, he believed that the disease began in a man after *skinning and eating of a tarabagan found dead on the hills*. He adds that these animals are very numerous in the parts visited by the epidemic. This was stated as well by former observers. Skrgivane<sup>(52)</sup> said that Preshevalski found in Northern Thibet a variety of the marmot called *Arctomys robustus*. According to the Thibetans this animal is also found in southern Thibet including Lhasa. Skrgivane adds

(46) Skrgivane, l.c., p. 606.

(47) Ibidem, p. 607.

(48) Russian Archive Pathol. etc., 1899, pp. 242-250.

(49) Vestn. Obst. Guig. 1909, pp. 897-909 and 1045-1039.

(50) China Med. Jl., March 1918, p. 146.

(51) Ibidem, p. 86.

(52) l.c.



the disease is well known in Thibet and it is due to the measures taken by the inhabitants that it does not assume an epidemic form. He reports that in the Chinese province Kansu, on the northern slopes of the Tian-Shan mountains both epizootics and human outbreaks are seen. This confirms the above statements of Parry. The importance of Thibet has already been emphasized by observers, e.g. (a) Rennie<sup>(53)</sup> who believed Thibet to be the possible source of the Yunnan and Mongolian outbreaks and (b) Koch<sup>(54)</sup> who pointed to Thibet as the real centre for the Chinese and Indian epidemics. Attention should be drawn to a fatal disease with buboes ("Beulenbildung") occurring from time to time in the valley of the Salwen river arising from Thibet and flowing through Burmah.<sup>(55)</sup> Mueller<sup>(56)</sup> who quotes this, adds that these reports point perhaps to the true origin of the Yunnan plague. It is quite natural that we should have no complete data for Thibet, which in a medical sense is still a *terra incognita*. These facts prove that there is no further reason to be sceptical about the presence of plague in that centrally situated country, as the German Plague Commission (1900) without sufficient evidence was obliged to be. Zabolotny<sup>(57)</sup> maintains that further proof of the existence of plague in Thibet is established by Paltshikovski's findings in Chinese Turkestan which confirm what was suspected by him before.

Before we continue to discuss individual endemic centres in Asia it would be well perhaps to look back. We have thus far seen three different sets of plague foci in Asia: (a) Assyr, (b) Western Asia with its centre in Kurdistan and (c) a larger area in Eastern Asia, which may be considered as one whole.

We are not able to find from available literature any connection between (a) and (b) and, as far as can be seen the endemic area in Assyr (Arabia) stands isolated by itself. Still the fact that this territory is connected by caravan routes with the centre of gravitation of the Moslem faith in Mecca, makes one suspect that plague was at some early period introduced into this country and had there gained a firm and lasting foothold.

We have to show now what connecting links lie between the Western and Eastern areas in Asia.

10. *Turkestan*. As just mentioned we have proof of the former existence of plague in this part of Central Asia. Sven

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(53) B. M. J. 1894, p. 615.

(54) D. Med Woch. 1898, No. 28.

(55) Kreitner, Petermann's Mitt., 27, p. 243.

(56) Die Pest, Wien 1900.

(57) Pestis bubonica, 1907, pp. 5-9.



Hedin<sup>(58)</sup> found in 1894 some traces of plague in the Tarim basin; he was warned not to visit Yarkand in summer, because it was constantly attacked by the pest during the warm months. In 1898 an outbreak was reported in Kishlah with 225 deaths out of 350 inhabitants. According to the Local Gov. Board Rep.<sup>(60)</sup> plague was recorded 1902 from four points in this region: (a) Barumsal in the province of Kashgar; (b) Kandshut in the same province; (c) Badaschan on the south-western slope of the Pamirs and (d) Schaschpal. Paltshikovski was sent to Barumsal and established the diagnosis of plague bacteriologically. The English Report<sup>(60)</sup> adds "it is believed that plague was brought by travellers from India across the Karakoram Range and the Hindu Kush." We have no further evidence to confirm or disprove this scanty bit of information, but must refer in addition to Zabolotny's statement regarding a connection of this plague area with Thibet and to the existence of the pest in western (Russian) Turkestan. A suspicious epidemic was reported in 1907 from the Atbaschinsk district, on the Aksai plateau.<sup>(61)</sup> This region is situated in the south-east of Russian Turkestan quite near the frontier of Chinese Turkestan. The origin of this outbreak is curious and as follows:— A Kirghese caught a *black marmot*, brought it in his jurte (tent) and skinned it. He fell sick soon afterwards and a localised outbreak of pneumonic plague affecting 46 persons arose, confirmed bacteriologically by Shendrikovski.<sup>(62)</sup> Another outbreak was recorded in July 1910 in the province of Semiretchinsk and in two villages of the Abbastin quarter in the Prjevalsk district of that province.<sup>(63)</sup> The last locality lies still farther east near the frontier of Chinese Turkestan.

In the districts west of Turkestan plague outbreaks have been known longer. Simpson mentions an outbreak in Samarkand and Bokhara already as early as 1056. Klodnitski<sup>(64)</sup> reported one outbreak in Afghanistan (1884), one at Merv (1885-87), two extensive ones in Astrabad, Meshed (1887) and finally one in Anzob (1898). He traced all these outbreaks to Khorassan. Plague appeared in September 1892 at Askabad.<sup>(65)</sup> In 1896 there was an epidemic further east in Merv, suspected to be plague. In 1898 finally there was the well known outbreak in the Hissar range south-east of Samarkand (167 versts). The Russian Government asserted that the infection was "im-

(58) Quoted by Sticker, *Die Pest*, Giessen 1908, p. 408.

(59) Mueller, *l.c.*

(60) Loc. Gov. Board Rep. 1902-03, p. 338.

(61) *Ibid.* 1907-08, p. 252.

(62) *Otchet Nar. Zdravitsa* 1907, p. 162.

(63) Manch. Pl. Serv. Rep. 1911-13, p. 17.

(64) Rep. Astrakh. Confer. 1910, p. 100.

(65) Loc. Gov. Board Rep. 1879-98, p. 246.



ported to Anzob through Baluchistan and Afghanistan from India by pilgrims who had returned from the pilgrimage to Mecca by way of Karachi, a port which was known to be infected. But no facts in support of this assertion have been brought forward." The report adds that it is not unlikely "that in the villages like Anzob in that district *there may be an epidemic like the "Mahamari" of Damaun (?) and Gurichal, which now and again comes in observation in the remote mountain villages.*" Levin<sup>(66)</sup> in a vivid description of the Anzob outbreak, appeared to regard the disease as endemic and laid much stress upon 30 odd cases of scars which he found in the inguinal regions of persons supposed to have recovered. In some instances these examined traced their sickness to 20 years previously. We think this point worth serious consideration particularly as the locality lies about half-way between the affected areas in West Turkestan and the established endemic area in Kashgar.

11. *Persia.* This country is perhaps not an endemic centre in the strict sense but nevertheless deserves our closest attention. It has been seen already that the outbreaks in the western parts of Persia can be traced to the common endemic centre in Kurdistan, but there are two more groups of outbreaks to be considered:

a) Those occurring since 1876-77<sup>(67)</sup> in the province of Khorassan. Mahé<sup>(68)</sup> *"regards this repeated appearance of plague in the N. E. of Persia as pointing to a possible relation between the plague centres of Mesopotamia and Khurdistan on the one hand, and those to the N. E. of the Himalayas on the other."* Proust<sup>(69)</sup> also expressed the opinion that *apparently the whole of the highland from the Caspian Sea to the Himalayas* had never been free from plague outbreaks. All the evidence collected by us points certainly in the same direction.

b) In 1905<sup>(70)</sup> Plague was reported from the province of Seistan, starting S. E. of Lake Helmund. Some tried to establish a connection between this and an infectious disease among cattle, which however was finally diagnosed as anthrax. Others thought of an importation from India through merchandise. A third theory was that "the malady had been prevalent among the nomadic tribes for a considerable time before it was recognised as plague and that the precise source of its

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(66) Vratich, 1893, p. 157.

(67) Loc. Gov. Board Rep. 1879-98, p. 209.

(68) Recueil de. Ccm. Consultat. d'Hyg. Publ., Vol. XI, p. 242.

(69) La defense de l'Europe contre la Peste, Paris 1897, p. 96.

(70) Loc. Gov. Board Rep. 1906-07, pp. 87-88.

Vetlianka 1878-79. There is however no doubt that plague infection was brought from Astrakhan by water fowls devouring plague sick rats and harbouring their fleas temporarily. Only the second and particularly the third causes seem to us possible; as for the third, we must emphasize the geographical position of this locality in relation to the plague areas in Russian Turkestan. It must be mentioned that no epizootic among rats was found, although fleas abounded. In Khorassan Grekoff succeeded in 1912 to prove an epizootic among field rats.<sup>(71)</sup>

12. *Astrakhan and adjoining territories.* Attention has been rivetted upon this area since the well known outbreak in Vetlianka 1878-79. There is however no doubt that plague had occurred in these parts for a long time. A good survey of this question is given in the Report of the Russian Plague Commission.<sup>(72)</sup> Outbreaks of "pestilence" were already recorded at the end of the 11th, in the 12th and 13th century, but they cannot be considered definitely as true plague. The first authenticated outbreak is that occurring at the time of the Black Death (1364). In the following centuries Astrakhan was repeatedly visited by plague, which according to the unanimous opinion of Russian authors was up to the 19th century always imported into the country and not of an indigenous nature. They believed it was introduced from the West, the only exception being the deadly epidemic of 1692-93. The origin of this visitation, killing over 10,000 out of 16,000 inhabitants in Astrakhan, is not clear, but it seems to have come from the Eastern foci.

While stating that in the 19th century the Astrakhan District became itself a plague focus, Russian observers seem rather inclined to connect the first outbreak of 1806-08 with the Caucasus, where pest had reigned from 1798 to 1828. Isaëff emphasized that the second outbreak in 1878-79 at Vetlianka was not imported but really endemic. He drew attention to cases of *pestis minor* occurring before the Vetlianka epidemic in the city of Astrakhan (1877) and pointed to a possible connection between these and the fatal plague in Resht (Persia, 1877).

It would be beyond the scope of this paper to enter into a detailed description of the long controversy *pro* and *contra* the endemicity of plague in those regions. Only a short outline of the problem can be given here. The Russian observers

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(71) Clemow, *Lancet* 1913, June 14, p. 1697.

(72) Papers of the Russian Plague Comm. Part II, edited by Isaëff, Petersburg 1907, p. 7 and foll.



contended that plague in these regions was imported and blamed the pilgrims, here as elsewhere, unnecessarily. For a time the camel was once suspected as a carrier of infection, but although its role was suspicious in a few limited outbreaks, this theory cannot be satisfactorily maintained.

The possible role of the hamster was considered too, but as far as we can see, no results were obtained, although a Commission was appointed to study this question.<sup>(73)</sup> Finally it was ascertained, that the actual carriers of plague here are two species of rodents (*spermophilus*, *Suslik*, and *Jerboa*).<sup>(74)</sup> Lately a third rodent, the wild mouse, was found to suffer also from plague epizootics and to be responsible for the epidemics in autumn and winter.<sup>(75)</sup>

Klodnitzki<sup>(76)</sup> referring to the endemic character of plague in the Kirghiz steppes, says that it is impossible to state when plague was introduced, whether at the time of the Vetlianka epidemic or even earlier. Equally difficult it seems to us, is the question *from where* the disease originated—the Western or the Eastern focus as Astrakhan and the adjoining affected territories lie, like Persia and Russian Turkestan, just between the two foci.

#### CONCLUSIONS.

It was the custom of former observers to fix certain definite localities as endemic foci of plague. As will have been seen from our review, it is difficult to draw any sharp line as to where one focus begins and another ends. So far as Asia is concerned, we may safely say that the whole of the central plateau is one huge endemic area. In the North we have recorded outbreaks in Transbaikalia; in the South at Kumaon and Gurwhal; in the East at Weichang and some parts of Inner Mongolia; in the West Kurdistan. The several outbreaks originating in so-called endemic foci, as claimed by various authors, are in our opinion only localised manifestations from one common source. In Central Asia the virus is constantly kept alive among the various species of susceptible rodents, which as we have seen suffer from periodical epizootics. Such visitations in their turn may result at one time or another in human cases.

The task of assigning the original role to any particular species of rodents is not easy, especially as the information

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(73) Bull. Office Internat. d'Hyg. Publ. 1913, pp. 1544-1551.

(74) Berdnikov Zentralblatt f. Bakt., 1913, pp. 251-259.

(75) Zaboletny, Arch. f. Schiff- u. Tropenhyg. XXVI, 1922, 12, p. 382.

(76) Astrakhan Conference 1910.

regarding them has not been uniformly worked out. Starting with the oldest known epidemic on record, such as that mentioned in the Bible, one may be tempted to lay the blame entirely upon Western Asia. It is questionable how far one is justified in considering this outbreak as really the first in history. Knowing as intimately as we do the habits of the tarabagan (Siberian marmot) and its close relationship to plague, we may perhaps be excused in assigning to it a principal role in its causation. It is quite possible that if other rodents, especially those marmot-like ones of Western Himalayas, etc., be similarly investigated, they may be found to play an equally important role, and thus help to confirm our view that the original home of the pest lies in that vast central Asian plateau, inhabited by these burrowing and hibernating animals.

This idea has been held by observers early in the present pandemic, like Le Dantec,<sup>(77)</sup> who said among other things that the tarabagan was the real cause of plague, and that it, not the rat, should be exterminated.

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## II. HISTORICAL EVIDENCE.

Turning now to the historical aspect of our subject, we find considerable evidence to support the above view. Let us discuss first the famous Black Death of the 14th century. The history of its origin, in spite of the voluminous attention devoted to it, is still obscure.

Some authors maintain that the disease was not a recent importation, but that it had been firmly entrenched in Europe, merely assuming pandemic features in the middle of the 14th century. Most of these authors think that plague was introduced into Europe by the returning Crusaders. It must be admitted that there was much epidemic disease and "pestilence" in the armies of the Crusaders; for instance, Gibbons alludes repeatedly to this fact. It can be proved also that plague was active in Europe from the 11th century onwards. A virulent epidemic raged there already in A.D. 1094, i.e., two years before the Crusades began. It would be wrong, however, to base any conclusions upon these facts alone; the evidence of the contemporary records leaves no doubt, that "the Black Death was in a very definite sense imported from Asia" (Payne).<sup>(78)</sup> While all contemporary and most modern writers agree upon this point, widely different views are held

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(77) *La Pathologie Exotique*.

(78) *St. Thomas's Hospital Reports*, vol. XVII.



as to where particularly the pandemic started. When we consider that rather strange theories are held even for the origin of modern outbreaks, in spite of an advanced knowledge of plague literature and of quick communications, we may realise more fully the difficulties of the ancient writers and form a fairer judgment of their testimony. Even nowadays the first seriously affected locality or the distributing centre is often regarded as the starting point. Is it any wonder then that this was done in Justinian's time (Pelusium) and similar conclusions were formed in the case of the Black Death? For this second pandemic China is frequently blamed (Payne). It is possible that epidemics raged there from 1333 or even earlier (Hecker),<sup>(79)</sup> though it is not easy to decide whether the "pestilences" of the old records were all plague. Creighton, for instance, believes<sup>(80)</sup> that plague was not prominent before 1352. Be this as it may there is no doubt that during the Black Death China was attacked secondarily. This view is advocated by the great authority of Hirsch<sup>(81)</sup> who says: "zum groessten Teil lauten die Angaben in Bezug auf die von der Krankheit zuerst ergriffenen Gegenden sehr unbestimmt oder beziehen sich auch nachweisbar auf erst spaeter befallene Gegenden (so namentlich auf China) ....." "Either the data regarding the first attacked districts are rather indefinite or regions said to be primarily affected were really invaded secondarily—especially China." Hirsch favoured the theory that the Black Death came from India; he relied upon the testimony of the Russian chroniclers and upon a statement of Fracastoro, who 200 years after the beginning of the epidemic, in a Latin poem "De syphilide," assumed the area of the Ganges river to be the original home of plague. Hirsch believes that pneumonic plague is a peculiar modification of the "Indian Strain," thus necessarily his attention is drawn towards India. In fact, our knowledge about plague in India in the 14th century is rather scanty. Nathan states: "Only two direct references have been traced which may point to the existence of plague in the West of India in the fourteenth and fifteenth centuries. The first is from Ibn Batuta, who notices that Muhammed Tughlak's army in Malabar (1325-1351) mostly perished of pestilence, and that at the end of the century (1399), after Timur left, the districts through which he had passed were visited by pestilence."

We believe that the attention of the contemporary writers was drawn to those two populous countries not because they were the starting points of the great pandemic but because

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(79) Hecker. *qu.* by Korsakov, Vj. Guig., 1900, p. 676.

(80) History of the Epidemics of Great Britain, 1891.

(81) Handb. der Hist.-Geogr. Pathol., Erlangen, 1860.

of their huge death toll. We think that both countries were infected from Inner Asia. The statements of many other recorders point in the same direction, so far as their limited geographical knowledge admitted.

The Russian Chroniclers (Richter,<sup>(82)</sup> Clemow<sup>(83)</sup>) asserted that one invasion into their country came from "India," i.e. from the South East. Nicephoras Gregoras<sup>(84)</sup> points to the Scythians, the Lake Maeotis and the mouth of the Don, districts well within reach by contiguous spread from Central Asia.

Gabriel de Mussis<sup>(85)</sup>, a Genoese lawyer, possibly an eye-witness of the disease in the Crimea, stated that "innumerable tribes of Tartars and Saracens perished in these regions by an inexplicable disease. Whole tracts of country, innumerable provinces, splendid kingdoms, cities, camps, and towns abounding in population, were attacked by a horrible death, and in a short time denuded of their inhabitants." It is to be noted that the Tartars were at that time engaged in warfare, besieging the Italian settlements. This makes it still more probable that the disease was brought from the East.

The Arabian writers, e.g. Ibn Wardy, said that the Black Death came from the "land of darkness," i.e. the kingdom of Kiptack with its capital Surai on the East of the Volga. Simpson<sup>(86)</sup> states that this part was considered by the Arabs as "nearly the limit of the habitable world." He remarks also that "Tartary," mentioned in the same respect by the Arabian historian, Mahassin, is a rather flexible term.

All available data seem to bear out Hecker's<sup>(87)</sup> opinion, that the Black Death started in the highlands of the Himalayas.

A new difficulty arises however when we consider how the disease reached Europe.

The Italian ships returning from Caffé (Theodosia) were neither the only nor the earliest instruments of importation (1348). Payne, for instance, mentions "two other ways by which the disease also approached, namely by way of Tiflis and Armenia into Asia Minor, and by the way of Mesopotamia and the Euphrates into Egypt." Constantinople, attacked

(82) *Geschichte der Medizin in Russland*. Moskau, 1813.

(83) *Ind. Med. Gaz.* Sept., Oct. 1898; *Practitioner*, Oct. 1894.

(84) Simpson, p. 21.

(81) Payne, *l.c.*

(86) p. 22.

(87) *Volkskr d. Mittelalters*, bearb. von Hirsch, Berlin 1865.



in 1347, was certainly one of the gateways. Both roads lead through the homestead of plague in Western Asia, old even at that time. Thus it is quite natural for the question to be raised by some authors,<sup>(88)</sup> whether the Black Death started from there or not. It seems to us that all the evidence points with great probability to Inner Asia as the source of the Black Death.

It is not our object to give a detailed analysis of the period intervening between the Black Death and the pandemic which started in 1894. Two points may nevertheless be accentuated to show that even during this comparatively quiet interval, there was a tendency for new waves to appear as if starting from one common focus. Thus (1) the epidemic in Cutch, Káthiáwár (India) occurred in 1812-21 at a time when the pest was equally prevalent in the Levant, spreading to the Lower Danube, Asia Minor, Armenia and Northern Africa, and lasting nearly 20 years.

(2) The Pali plague (India) of 1836-38 corresponded in time with a fresh and comparatively limited activity in the Levant affecting Turkish Dominions and Egypt. In Rajputanā the invasion disappeared at the same time as in the Levant.<sup>(89)</sup>

In viewing the pandemic which began in 1894 and was distributed mainly through the busy sea-port of Hongkong, we find again plenty of evidence to show its tendency to radiate from a central focus: for instance,

- (1) In 1899 the disease was unusually extensive and virulent in Outer Mongolia, being recorded in three separate regions and killing over 400 persons. The pneumonic type predominated.
- (2) Plague was seen to assume a more active form in Astrakhan about the same time that it invaded India in 1896.<sup>(90)</sup>
- (3) In September 1899 the Russian authorities at St. Petersburg were alarmed "at the increasing ravages of a form of "Malarial fever," and spreading over a large area in certain parts of Central Asia. Plague had on some previous occasions been designated elsewhere by the name of epidemic malarial fever."<sup>(91)</sup>

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(88) Simpson, p. 22.

(89) Simpson *l.c.*, pp. 40-47.

(90) Klednitzki. Astrakhan Conference 1910.

(91) Loc. Gov. Board Rep. 1898-01, p. 123.

## III. CONCLUDING REMARKS.

In conclusion I may say at once that I am not trying to bring forward any new theory. The material for this article has been largely gathered from the work of past authors. At the same time I cannot help feeling that recent workers have been too much engrossed in certain limited foci without paying sufficient attention to their mutual relation to one another. The role of domestic rodents in the epidemiology of plague has been confirmed more than once, but this is the first occasion on which the part played by the tarabagan has been thoroughly worked out. It is possible also that the original virus may rest in the wild rather than in the domestic rodents. In the same way that Central Asia is now generally regarded as the cradle of the human race, as it assuredly is the first habitation of the wild rodents, so we may say, that the original home of plague was also situated in these regions.

WU LIEN-TEH.



# 圖明說域區疫鼠有固

MAP TO ILLUSTRATE ENDEMIC PLAGUE AREAS.

## GUIDE TO MAP.

- 1a. Benghazi, Tripolitania
- 1b. Morocco
- 2a. Victoria Nyanza
- 2b. Mauritius and Reunion
- 3. Assyr
- 4. Khurdistan
- 5. Kumaon and Gurwhal
- 6. Yunnan
- 7a. Transbaikalia
- 7b. Outer Mongolia
- 8. Inner Mongolia
- 9. Thibet
- 10a. Chinese Turkestan
- 10b. Semiretchinsk
- 10c. Russian Turkestan
- 11. Persia
- 11a. Khorassan.
- 11b. Seistan.
- 12. Astrakhan.







## REMARKS ON THE INCIDENCE OF CERTAIN DISEASES IN CHINESE AND EUROPEANS.

*(Read before the Conference of National Medical  
Association, Feb. 1924.)*

Racial partiality to certain diseases, such as the prevalence of diabetes among the Jews,<sup>(1)</sup> is thoroughly well known and recognised. Most of us who see much of the sickness among the Chinese cannot help observing that there are certain diseases or features of a particular disease which are frequently met with, but which are rare in England or America. Conversely, there are common European diseases which are but seldom seen in the Chinese patients. We are not of course, referring to tropical diseases, but to those common to the Oriental and Occidental.

In introducing this vast and interesting subject, my object is to arouse discussion and to start investigation and research. At the present moment, very little is known about some of these peculiarities. For instance, why is appendicitis comparatively rare among the Chinese and why is fistula-in-ano so common?

I propose to append two lists which may have bearing on the title of this paper. The lists are necessarily and absolutely incomplete, though they have been compiled from several hospital reports, and text books.

### FISTULA-IN-ANO.

Among the commonest conditions is fistula-in-ano. Libbey<sup>(2)</sup> stated that he saw large numbers of the disease in Wuhu and operated on 72 cases (among 455 operations) in one and half years, that is to say 16%. As many of these patients were farmers and boatmen, he attributed the etiology to intestinal parasites. The most likely explanation is constipation and coarse food. The bowel is relieved only when absolutely necessary. The hard stool and/or the coarse constituents tend

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(1) The Principles and Practice of Medicine, Osler, p. 430.

(2) Fistula in ano, W. E. Libbey, China Medical Journal, Vol. XXXV, No. 1, page 18, Jan. 1921.

to cause abrasion of the mucosa of the rectum and so favouring infection by bacteria. An ischio-rectal abscess is formed and a fistula resulted. However, there are other cases which are tuberculous in origin; to what extent it is unknown.

### TUBERCULOSIS.

Tuberculosis is wide-spread and we see much of it, very often in the last stages. In dealing with tuberculosis of the bones and joints, one is obliged sometimes to amputate the affected limb. The results<sup>(3)</sup> of the use of sun-light and carbon arc light lamp in the treatment of lupus and other forms of tuberculosis tempt one to utilize the bright sun-shine of Manchuria. But alas, the time factor is a stumbling block. The average hospital patient is a poor man and wants to get well quickly, so as to be able to earn some money. Moreover, there are many medicine-men in the city who promise to cure very quickly, and so it is difficult to give the treatment a fair trial, though we practise it as much as possible. When we encounter a cold abscess, or softened gland, we withdraw the pus by a needle and inject the iodoform, creosote, ether and olive oil mixture at weekly intervals. This procedure also takes time and sometimes 6-10 injections are required, but the results are gratifying. During the last 10 months, we saw 1705 cases of tuberculosis in the Harbin out-patient department, as follows:—

Glands .....	748	43.8%
Skin ....	312	18.3%
Bones .....	257	18.0%
Lungs .....	196	10.9%
Joints .....	133	7.8%
		<hr/> 98.8%

It is interesting to note that the glands come first, then the skin, and then the bones. As the Chinese do not drink milk, the source of tubercle must be human.

In their small, ill-ventilated sleeping quarters, a person with lung tubercle or open tuberculous sores will easily infect his room-mates. The tubercle bacilli, spreading either by droplet infection or in the form of dust, are inhaled and deposited mostly on the mucous membrane of the nose and mouth. The lymphatic system draining these areas has to deal with this invasion, hence we see so many T. B. glands.

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(3) British Medical Journal, Sept. 1923, No. 3273, page 493.



We have to note that the lung takes the fourth place in frequency.

### MALIGNANT SCARLET FEVER.

Scarlet fever in Europe is not such a dangerous disease; many children are attacked but they recover. The mortality is said to be 3%. In China, this disease was unknown previous to 1873, and as is the way of new diseases, it developed into a virulent type. Many Chinese children die of it in the Treaty Ports. An epidemic of scarlet fever appeared in Harbin in the autumn and winter of 1923 with 10-20 cases reported weekly. One family lost all the three children. It is presumed that the Chinese have not as yet developed an immunity against this disease. When the foreigner is infected in China, he is apt to get a severe and sometimes fatal attack, the case-fatality among 68 foreign patients admitted to the Shanghai Isolation Hospital from 1905 to 1916 was 15.4%.

### SYPHILITIC RHEUMATISM.

We have met with many cases of syphilitic rheumatism, but few cases of locomotor ataxia.

For the last ten months, 689 cases of syphilis were seen in our out-patient department:—

1st stage .....	125	18.00%
2nd ,, .....	489	70.97%
3rd ,, .....	75	10.88%
4th ,, .....	0	0
		—
		99.85%

This great preponderance of the secondary stage has not been sufficiently emphasized, nor has the prevalence of syphilitic rheumatism been recognised. The usual explanations set forth to account for the rarity of locomotor ataxia seem inadequate, while no explanation at all can be given for the frequency of syphilitic rheumatism. We had 1490 visits from syphilitic rheumatic patients, while not one locomotor ataxia or general paralysis of the insane has been seen, during the last four years.

Dr. A. H. Woods<sup>(4)</sup> of the Canton Hospital reported that he found one tabetic case amongst 175 neurological patients; and one amongst 472 patients with general diseases.

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(4) China Medical Journal, March 1918, Vol. XXXII, No. 2, page 113.

## APPENDICITIS.

We have admitted two cases of appendicitis, during the last four years. Other hospitals report higher admissions, and it appears as if this condition is not so very rare as it was thought to be at one time. However, from all accounts, the Chinese do not suffer from it so frequently as the Europeans.

In the Harvard Medical School reports, it is interesting to compare the figures, as Chinese and Europeans are admitted into the same hospital.

Appendicitis cases among Europeans—20 cases in	
240 admissions .....	8.3%
Appendicitis cases among Chinese—10 cases in 766	
admissions .....	1.3%

From what one can gather, it seems the patients are seen, as a rule, in a late stage, possibly the abscess or faecal fistula stage. It is possible that with the gradual adoption of the European mode of living and style of food, appendicitis will be seen more frequently than at present.

Also, if the patients seek Western medical advice earlier, and if the surgeons are more enterprising, the records of appendectomy at a favourable period will be higher.

## CARCINOMA.

Carcinoma is not really so rare in China as they would have one believe. From the appended list, it can be seen it is often met with. Numerous factors, however, prevent the surgeon from seeing or operating on the sufferer.

Still, it can be assumed that it is rare when compared to European figures.

Again it is interesting to speculate the real reason for this difference.

Dr. Dyce Sharp<sup>(5)</sup> writing to the British Medical Journal remarked that the negroes in West Africa very rarely suffer from cancer, or appendicitis. Sir William Osler stated that it is exceedingly uncommon to find a case of locomotor ataxia or chorea among the negroes. Is it, then, the simple and primitive life that the negroes lead that render them immune to these diseases?

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(5) British Medical Journal, July 1923, No. 3263, page 86.



## CHLOROFORM AS AN ANAESTHETIC.

We have only one death from chloroform during the last eight years. When I was in Shanghai, acting as a resident surgeon to the Shantung Road Hospital for two years, we had one case. Dr. So To Ming of the Canton Hospital administered chloroform to over 10,000 patients during a period of 3 decades without a death.

The Chinese patient takes this anaesthetic very well. They are not nervous or frightened, but breathe quietly and "go under." After waking up, they do not suffer from post-anaesthetic complications, but recover quite quickly, sometimes in a surprising manner.

In England, the condition of things is quite different. In spite of the skilful anaesthetists, deaths from chloroform are frequently met with. In a large London Hospital 42 deaths from chloroform were recorded in 8 years, it is said.

Before closing this subject, it is only fair to state that here in China we often pick our patients for operation, and most of the patients are in a robust and healthy condition (young labourers). Still, apart from these considerations, one cannot help remarking on the ease and safety with which the Chinese patient takes chloroform anaesthesia.

## CONCLUSION.

One is constantly struck with the entirely different kind of hospital practice in China. In London I used to assist in operations such as for appendicitis, hernia, varicose veins, varicocele, tonsils and adenoids, acute abdomens, and other major operations. But in China, I have quite another type of operations to perform. We have to deal with more surgical than medical conditions, and in the majority of cases, the disease is seen in a late stage, what is aptly termed "ultimate pathology." In our out-patient department, we see large numbers of septic skin affections, tuberculosis in all forms and locations, syphilis and affections of the alimentary canal. The great prevalence of these diseases may be explained by the lack of personal hygiene and the absence of modern public health measures. On the other hand, the rarity of certain diseases may be accounted for by the simple life, sensible clothes and plain food of the people.

Further, the fact that western medicine is a recent introduction into this country may have some effect on the kind and stage of the diseases admitted into hospitals.

However, some of the points brought up can hardly be accounted for in some such manner.

If this paper has succeeded in stimulating interest and enquiry into this absorbing subject, it will not have been written in vain.

DR. J. W. H. CHUN,  
*Senior Medical Officer, Harbin.*



## A.—Diseases which are comparatively common among the Chinese.

	Plague Prevention Service Hospitals 1918-1922 Admissions 1325	Shantung Road Hospital, Shanghai 1914-1922 Admissions 19219	Red Cross Hospital, Soochow 1917 Admissions 1898	Union Medical College, Peking 1916-1917 Admissions 1199	Temple Hill Hospital, Chefoo 1916 Admissions 332	Red Cross Hospital, Shanghai 1914-1917 Admissions 986	Diseases of China, Jeffreys and Maxwell	Shanghai Health Officer's Report May 1918
Fistula in ano	78	338	2	58	13	16	One of the commonest Surgical Conditions	
Soft Fibroma	15	26		4	0	2	Very common and large	
Low mortality in influenza	1 in 24			0 in 3				The mortality is likely to be very low
Low mortality in typhus	2 in 56						Mortality 10 to 20%	
Low mortality in Small-pox	1 in 9							
Syphilitic Rheumatism	28							
Piles	26	119	20	23	14	8	Exceedingly Common	

### B.—Diseases which are comparatively rare among the Chinese.

	Plague Preven- tion Hospitals 1918-1922	Shantung Road Hospital, Shanghai 1914-1922	Red Cross Hospital, Soochow 1917	Union Medical College, Peking 1916-1917	Temple Hill Hospital, Chefoo 1916	Red Cross Hospital, Shanghai 1914-1917	Diseases of China, Jeffreys & Maxwell	Principle of Medicine, W. Oster,
	Admissions 1325	Admissions 19219	Admissions 1898	Admissions 1199	Admissions 332	Admissions 986		American figures
Appendicitis	2	29	27	11	1	10	I in 2000	% 0.05
Tabes dorsalis	0	0	0	0	0	0	O in 12000	% 1.2
Carcinoma	7	41	10	27	8	15	External com- mon internal rare	201 in 16562 cases
Death from chloroform	0	none recorded	0	0	0		Exceedingly rare	
Sprue	0	0	0	0	0	0	Rare	
Varicocele	0	0	0	1	0	0	Rare	
Diabetes	(1)	0	0	0	0	2	Few recorded	276 in 27618 Admissions
Rickets	0	0	0	0	0	0	Rare	50 % to 80 % of Children
Rheumatic Fever	0	8	0	7	1	1	Rare	285 in 9286 Admissions
Hernia	12	73	9	16	2	8	Very common	
Chorea	0	0	0	0	0	0	None	
Hysteria	0	4	0	3	0	0	None	
Pes Planus	0	1	0	2	0	0	Not mentioned	

(1) 85 Cases seen in 91,000 out-patients=0.09%

(2) 1 Case seen in Out-patients Dept.



## SCARLET FEVER IN CHINA.

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## A. INTRODUCTION.

It is generally understood that Scarlet Fever is a disease of temperate climates, being almost unknown in the tropics and seldom seen in subtropical countries. As China (including Manchuria) lies between  $20^{\circ}$  lat. in the south (Hainan) and  $54^{\circ}$  lat. in the north (Taheiho, Heilungkiang), great variations may be expected in the incidence of this widespread communicable disease. With a view to ascertaining the exact prevalence of Scarlatina throughout the country, a *questionnaire* was sent to different medical friends from Canton in the south to Aigun in the north asking for details regarding the following:—

- a. Existence or not. Morbidity and mortality.
- b. First appearance in the locality.
- c. Sex and age incidence.
- d. Comparison between Chinese and foreigners.
- e. Any unusual features.

The replies received are not all uniform in character, but enough information is gleaned to enable us to form a fairly

accurate idea of its nature in different parts. To supplement these statements, the files of the China Medical Journal, Customs Reports and all available medical publications are consulted. It will be seen that considerable gaps in the reports exist, but it is hoped that these data may stimulate others to study the question in their respective fields and thus help to explain among other things the reason for its unusual virulence in certain regions and even at different times in the same region. Scarlatina is believed to be a recent importation into China and Japan, and its comparative frequency among white people of the south where few if any Chinese cases are seen is an interesting phenomenon. W. G. Lennox, in a statistical study of the health of foreign missionary families in China (1300 marriages and 3254 children) came to the following conclusions:—

- a. Scarlatina, like other exanthematica, is contracted less frequently inside than outside China.
- b. Out of 193 cases among their children, 104 occurred in North China, 85 in Central China and 4 in South China. Of these 12.5% died in North China, 2.3% in Central China and none in South China.
- c. Altogether 16 deaths from Scarlatina occurred among 3254 children born.
- d. 53 adult missionaries took the disease, making a percentage of 3.4.

#### B. SCARLET FEVER IN HONGKONG.

Year	Population	Deaths	Plague	Small-pox	Scarlet.	Total Infectious Diseases
1904	—	—	510	—	1 (1E, 1C)	758
1905	—	—	304	—	1	598
1906	326,961	8379	893	192	1 (C)	1179
1907	—	7286	240	341	—	—
1908	336,448	9271	1073	472	3	1668
1911	456,739	7748	269	272	1	702
1912	467,777	9682	1847	709	8	2757
1913	489,114	8435	408	111	3	1913
1914	501,304	9585	2146	110	1	2521
1915	509,160	7921	144	34	0	507
1916	529,010	10558	39	712	2 (E)	1110
1917	—	10433	38	595	3 (E)	919
1918	561,500	13714	266	32	? 3 (2E, 1C)	1913
1919	—	11647	464	27	7 (2E, 5C)	1011
1920	648,150	12419	138	34	3 (2E )	560
1921	585,880	11880	150	191	1 (E)	763
1922	662,200	14569	1181	212	5 (E)	1717
		128591	7090	3039	41	15493



The rarity of Scarlet Fever in Hongkong can be judged from the above figures. During 12 years (1911-1922), when full records were obtainable, out of 15,493 cases of infectious diseases, only 41 were Scarlet Fever and of these 10 were among Europeans. During the same period 7,090 cases of bubonic plague and 3,039 of Small-pox were encountered.

### C. KWANGTUNG.

No municipal records are kept, but individual doctors have written, almost all to the effect that Scarlet Fever is exceedingly rare among Chinese. For instance:—

Dr. W. G. Reynolds and W. W. Cadbury have seen only two uncertain cases (one severe and one mild), both among foreigners, since 1916. The former occurred in a Portuguese boy of 8 years, infected from Hongkong. Dr. P. J. Todd, who has been in practice for 21 years in the city, has seen only one suspicious case, but he rather doubts its diagnosis.

Dr. Chas Selden, who has charge of the Chinese Mental Hospital accomodating 700 patients, has not encountered a single case of Scarlatina among its inmates. The only patient he saw was that of a foreigner in Macao in 1898, where the temp. was 102.5 and desquamation lasted for 8 weeks.

Dr. S. P. Nye, a Chinese physician who has been in practice for 30 years, has encountered no case.

Dr. Hans Kummels, a German physician with a large practice among wealthy Chinese, reports 'a very small number' of cases.

Dr. J. A. Hofmann, for the Hackett Hospital for Women says no cases have been seen.

Dr. J. M. Margaret, for the staff of Canton Hospital, reports no cases since the last decade.

Dr. Casabianca, in charge of the Doumer (French) Hospital, can only recollect one case—that of a Chinese student (native of Kwangsi Prov.) in 1919. "The symptoms were indubitable and complete with no complications and the evolution was altogether mild." Dr. Casabianca considers the disease quite rare. He has been in practice in Canton for 15 years.

Dr. E. W. Kirk of the New Zealand Mission Hospital, Kongchuen, Canton, says that since coming to Canton in 1910 he cannot recall having seen any case of Scarlet Fever in Kwangtung.

## D. CHEKIANG.

*Ningpo.* From the Customs Surgeon's Report April 1st 1907 to May 31st 1909 we find only one case of Scarlet Fever, contracted at another port. In 1918-19 Report, occurs the following:—

“ A particularly malignant type of scarlet fever rampant in this section. Cases received into the Hospital usually recovered under the serum treatment, but those seen in their homes invariably died.”

*Hangchow.* Report for 1912 mentions foreign cases of Scarlet Fever without details.

*Wenchow.* Is regarded as the dividing line between tropical and temperate China, thus separating a southern non-scarlet from a northern scarlet-producing zone. This arbitrary division is only roughly accurate.

## E. KIANGSU.

*Shanghai.* An excellent review is given by Dr. Arthur Stanley (then Principal Health Officer of the Settlement) in his report of 1917, and is herewith reproduced:

After a period of comparative absence, Scarlet Fever has become prevalent during the spring of the year, reaching almost epidemic proportions among certain sections of the population. The incidence, compared with what may be termed the initial epidemic of 1902, since when the foreign population has trebled and the Chinese doubled itself, was considerably less, say, about half the incidence of the 1902 outbreak. No special cause can be assigned except the increase of non-immune material. It may be taken that the majority of persons are to some extent naturally immune to Scarlet Fever but that during periods of comparative absence of the disease the reservoir of non-immune persons gradually fills up, so that the introduction of a sufficient quantity of infection determines the onset of an epidemic which lasts until the excess of non-immune persons is removed.

The first recorded death from Scarlet Fever in the Foreign Settlement of Shanghai was in 1873. It seems probable that the infection was imported. At about this time, cases are believed to have been reported at Chefoo. The occurrence of Scarlet Fever in Japan appears to have been officially notified in 1897, but it is probable that a few cases occurred prior to this. Indeed, Scarlet Fever



appears to have reached Japan and China at about the same time and to have been previously unknown.

By the year 1902 there had been introduced into Shanghai a quantity of infection sufficient to gather epidemic momentum, and the Chinese death record from Scarlet Fever in that year, 1,500, does not appear to have been exaggerated.

As would be expected with a recently introduced disease, against which evolution has afforded no natural immunity, Scarlet Fever has been of virulent type among the Chinese. It is probable that the passage of the disease through the susceptible Chinese has led to an intensification of the virus, so that it is more fatal to foreigners also. The average case-fatality among 68 foreign cases admitted to the Isolation Hospital from 1905 to 1917 was 18.2%. The general case-fatality of Scarlet Fever in England was in corresponding years below 5. The tendency in the home countries is for the type to be less virulent, with a case-fatality approximating to 3%. But Scarlet Fever is characterised by an exceptional variation and in epidemics the case-fatality may vary from 30% to nothing. In Shanghai there is no indication yet of any general diminution in virulence of type among either foreign or Chinese cases. On the other hand, in Japan the fatality appears to be reverting to the English and American type of low severity.

There have been in Shanghai curious groups of cases of 'mild sore throat,' without the characteristic signs of Scarlet Fever, accompanying typical cases. Such 'mild sore throats' appear to have occasionally carried the genuine infection and produced typical cases of Scarlet Fever. Some of these must be regarded as atypical cases of Scarlet Fever. Others as cases of tonsillitis, septic, follicular and catarrhal, are often accompanied by an evanescent eruption which does not desquaminate; and so common as to be likely to be usual accompaniment of outbreaks of Scarlet Fever and difficult, sometimes impossible, to distinguish from cases of atypical Scarlet Fever. This points to the necessity of isolating all 'sore throats' during an outbreak of Scarlet Fever of virulent type; but to keep cases not definitely diagnosed as Scarlet Fever separate from typical cases. As pathogenic organisms may be considered as true to type there seems to be no good reason for thinking that ordinary tonsillitis may develop into Scarlet Fever, nor that throats susceptible to tonsillitis are *ipso-facto* susceptible to Scarlet Fever.

In Shanghai there is a tendency towards quinquennial periodicity. The big initial epidemic of 1902 was immediately followed by four years of remarkable absence or reduction of cases; and then a moderate outbreak in 1907, gathering momentum again to maxima in 1912 and 1917. Especially after the initial outbreak in 1902 it would appear as if all the most susceptible material had been exhausted and that a new generation of young children was required before any further great devastation was possible. Above the age of five susceptibility to infection is generally held to become progressively less. As regards seasonal prevalence the incidence in Shanghai corresponds to the American type, where it is at its maximum in the spring and at its minimum in the fall; whereas in England the seasonal variation is just the reverse. The same seasonal variation occurs in Japan as in China. This may be accounted for by the hotter summer in those countries; anything approaching tropical heat appearing to be antagonistic to the propagation of Scarlet Fever, which is rare anywhere in the tropics and, when introduced, does not seem to be able to retain a hold. In Shanghai, although the maximum incidence is in March, cases may occur throughout the summer; even in July and August with a mean temperature of 80 F.

Scarlet Fever now appears to be pretty generally prevalent in Japan, though the figures are comparatively small. The comparative fatality of the infectious diseases in Japan roughly have the following order in general mortality figures during recent years—Typhoid Fever, Dysentery, Cholera, Small-pox, Plague and then Scarlet Fever. Scarlet Fever also occurs in the larger towns of Korea.

In China, Scarlet Fever seems to be generally prevalent in the Yang-tse valley, at least as far up as Hankow: also in the North in Chefoo, Tientsin, Peking and Manchuria. In Hongkong four cases of Scarlet Fever were reported to have been brought in by two British warships from England in 1898 and that the disease had previously been 'practically unknown' in the Colony. From 1908 to 1916 fifteen cases were notified. In Singapore, Penang, and the Philippines Scarlet Fever does not appear to come into the picture so far as health statistics show.



## INCIDENCE OF SCARLET FEVER REVISED TO 1922.

FOREIGN SETTLEMENT OF SHANGHAI									
	Foreign cases Notified	ISOLATION HOSPITAL						TOTAL DEATHS	
		FOREIGN			CHINESE			Foreign	Chinese
		Cases	Deaths	Case Fatality	Cases	Deaths	Case Fatality		
1873	...	(Foreign cases prior to 1905 isolated in General Hospital)	...	...	...	...	...	1	Prior to 1902 death figures not regarded as sufficiently accurate to quote; but it may be assumed that Scarlet fever was either entirely absent or sporadic in its incidence.
1882	...		...	...	...	...	...	2	
1888	...		...	...	...	...	...	2	
1889	...		...	...	...	...	...	2	
1893	...		...	...	...	...	...	2	
1897	4		...	...	...	...	...	0	
1898	8		...	...	...	...	...	0	
1899	7		...	...	...	...	...	0	
1900	15		...	...	...	...	...	3	
1901	58		...	...	...	...	...	11	
1902	101		...	...	34	7	21	27	1500
1903	6		...	...	7	0	0	1	2
1904	15		...	...	11	2	18	3	0
1905	5	11	0	0	1	0	0	1	0
1906	10	20	2	10	2	1	0	3	5
1907	58	70	12	17	43	7	16	14	79
1908	25	19	2	10	17	6	35	2	33
1909	9	7	2	29	16	2	12	3	9
1910	32	35	6	17	31	9	29	7	109
1911	15	22	3	14	25	7	28	2	35
1912	49	64	14	22	93	34	36	11	146
1913	32	56	11	20	113	32	28	15	115
1914	24	42	4	9	106	31	29	5	144
1915	15	38	1	3	89	22	25	1	147
1916	27	57	11	19	99	21	21	9	234
1917	113	153	40	26	209	50	24	38	595
1918	33	58	6	10.3	54	8	14.8	7	142
1919	19	23	1	4.3	22	4	18.2	1	86
1920	29	43	0	0	39	8	20.5	0	103
1921	28	43	3	7	60	19	31.6	3	149
1922	27	*						3	149
	764	761	118	15.5	1071	270	25.2	179	3782

\* F. and C. 160 cases (28 deaths) 17.5%.

## NOTES OF THIS TABLE:—

1. Incomplete notification reduces the value of the first column.
2. Decimal points omitted from case-fatality percentages.
3. Total foreign death figures in Shanghai may be regarded as the most accurate of the data available.

4. The higher case-fatality among Chinese cases in Isolation Hospital does not necessarily indicate a higher virulence of type or lessened resistance among the Chinese as compared with foreigners, for many Chinese cases are brought in moribund after outside treatment had failed.
5. The discrepancy between the foreign deaths in Isolation Hospital and the total deaths is accounted for by cases from outside the Settlement being also admitted.

*Chinkiang.* For the six months ending September 30, 1910, we read: "Scarlet Fever during the whole year. Attended four cases, notified six. Sporadic cases occurring in summer is unusual; writer laid the blame on the refugee camps, where he actually found scarlatina."

The reports of April-September 1914 state: "Scarlet Fever and Diphtheria have again occurred freely among Chinese, but have not met with foreign cases." In the next year: "Scarlet Fever has been somewhat prevalent, and one case (slight), which was however followed by very profuse desquamation, occurred in the Concesion."

In 1915-16, "Scarlet Fever has not been in evidence above the average."

In 1918: "several cases were seen among Chinese, none among foreigners."

*Soochow.* The reports state that in 1916 the usual number of scarlet fever cases were seen among Chinese. In 1918, the disease appeared to have been endemic during the whole year.

*Nanking.* The University Report states that in 1917 scarlet fever was very prevalent in the spring and winter months.

*Hsuchow.* In spring 1918, scarlet fever was very prevalent in a severe form, sweeping whole towns and hamlets.

#### F. SHANTUNG.

*Tsingtao.* We are unable to procure the reports of the Health Department which will surely be interesting.

*Taikuhsien.* In 1920, thousands were attacked by scarlet fever and diphtheria.

*Chefoo.* Hogg relates a mild epidemic in the school of the China Inland Mission for foreign children, evidently imported by steamer. No deaths resulted. (C.M.J. 1918 p. 239).



## G. KIANGSI.

*Kiukiang.* In 1912, two cases were recorded as having been imported from Shanghai.

## H. HUPEH.

*Hankow.* Dr. Robert Aird has kindly supplied the following information regarding this district, and refers us particularly to a considerable outbreak in 1917 mentioned in the Consular Medical Report for year 1916-1917.—

“Some anxiety was caused by the undoubted presence of Scarlet Fever among the Chinese population during the spring months (1917), and steps taken by the Municipality by means of leaflets in English and Chinese, to bring home to residents the need for care in order to prevent the spread of infection. The actual extent of the prevalence of the infection in the native city and in the neighbouring cities of Wuchang and Hanyang, was difficult to gauge as cases of diphtheria and of simple angina were also occurring about the same time. That the scarlet fever was of a malignant type was shown by the cases actually met with. Thus a Chinese mother and child were brought into the Roman Catholic Mission Hospital one evening, with bad throats and severe rash, and both died within 24 hours. A house-coolie living in the British Municipal buildings was taken ill and died within four days. A compradore's child in the German Concession was taken ill and recovered, but the compradore caught the infection, and died after a very few days' illness. From a patient in the London Mission Hospital the English nurse in charge unfortunately contracted the infection and developed a very severe attack, and died in five days. The presence of this deadly disease in the midst of a great Chinese population, new to it and without any acquired immunity constitutes a very grave problem, especially in view of the long period during which the disease is contagious, the over-crowding of Chinese dwellings, and the impossibility of adequate isolation and disinfection. It emphasises the undesirability of crowding Chinese tenement dwellings within the narrow confines of the Concession, for if the infection became at all prevalent in them the foreign population would be almost certain to suffer severely also, especially the children, who are more susceptible than adults.”

I have been in practice in Hankow since March 1904, and the epidemic above mentioned was certainly the most severe we have had during these twenty years, but sporadic cases turn up nearly every winter. We do not see many cases among Chinese, but those seen in foreigners can generally

be traced to a Chinese source of infection, e.g. a Russian child in a good home contracted the disease about a year ago, and it was subsequently found that a jobbing tailor who had been working in the house, had had his own child ill with the disease at home.

1. Whether the above epidemic was the first appearance of Scarlet Fever in Hankow or not, I am not quite certain, but I rather think it was not. I remember before that time a British child contracting the disease a couple of days after its father returned from a visit to Shanghai, but there were no subsequent cases of infection from this child.
2. Tables of Scarlet Fever as compared with other communicable disease I am sorry I have not got. Hankow has so many different authorities in its various Concessions, and native city, that there is no central organisation for the collection of such data.
3. Comparison between foreigners and Chinese is difficult owing to the absence of statistics, but personally I have seen more cases among foreigners than among Chinese.
4. Severity among Chinese and Foreigners? Those cases which I have seen among Chinese were more severe than those among foreigners, but I have not seen enough to be able to express much of an opinion on this point.
5. Scarlet Fever increasing or decreasing? I should say, neither. It occurs irregularly, usually during the colder months.
6. Case charts. I am sorry I have none on hand. My partner Dr. Skinner tells me a case he had last spring in a young Englishwoman showed a typical chart. The Russian child whom I attended last winter had also a characteristic temperature curve, somewhat prolonged by a persistent streptococcal infection of the nose.

*Wuchang.* Scarlet Fever was rampant in this centre in 1918.

*Ichang.* In the Report for 1916-17, it is recorded that "scarlet fever which was so prevalent in the lower river ports did not reach Ichang."

#### I. KWEICHOW.

*Kweichow.* A few cases were reported here in 1918.



## J. KANSU.

*Lanchowfu.* In 1919: "there are epidemics occasionally of.....scarlet fever. It is surprising that it should be regarded as a disease newly imported to China. I have seen it both in Chinese and foreigners in far inland provinces. Sometimes we seem to have it in very malignant form. (King, C.M.J. 1919 p. 38.)

*Sining.* This fever never leaves us. Every autumn and winter finds some children suffering, but every few years it appears among us in a more malignant form and cuts off children in large numbers. The malignant form has been prevalent this winter and hundreds of children have died. In one village of forty families, 60 children died. (C.M.J. 1916, p. 392).

## K. CHIHILI.

*Peking.* The Government Infectious Diseases Hospital was established in 1915 in the northern section of the capital. We have not been able to procure any published statistics, but Dr. Yen Chi-chung, the Director, has been kind enough to supply the following private information in form of a table.

Year	Sex		Total	Cured	% Cured	Deaths	% Deaths	Left before time
1915	7M	8F	15	6	40.0	9	60.0	0
1916	110M	94F	204	152	75.0	35	17.0	17
1917	38M	30F	68	46	68.0	17	25.0	5
1918	4M	4F	8	6	75.0	1	12.50	1
1919	4M	0F	4	4	100.0	0	0	0
1920	8M	6F	14	12	86.0	1	7.0	1
1921	41M	41F	82	59	72.0	19	22.0	4
1922	89M	59F	148	109	73.0	33	23.0	6
1923	51M	44F	95	77	81.0	18	18.9	0
	352M	286F	638	471	73.8	133	20.8	5.3%

Private practitioners also report a large number of cases every year, sometimes mild but often fatal. The latter are usually of the septic type, showing marked vomiting, high fever, headache, red tongue, swollen throat, etc. Complications, like adenitis, otitis, nephritis, rhinitis, are quite common, but rheumatism, myocarditis or endocarditis is rarely encountered. Scarlet Fever, because of its newness and withal virulence among the community, is much feared by the well-to-do classes, but so far insufficient educational work has been done in the way of communal hygiene, and the disease has lately been allowed to affect all classes.

*Tientsin.* For nearly fourteen years, we have observed very severe cases in this city among both wealthy and poor people. Owing to the widespread ignorance and superstition of the womenfolk, even after having undergone a general education, infection generally spreads from child to child in the same family until almost every one is attacked. The constant communication between the children and servants of the locality also helps to spread the disease broadcast. Even adults are often attacked as the following cases show:—

- a.* An experienced Chinese old style physician aged 62 (whose son is one of our medical officers) attended in Feb. 1923 the family of the late Gen. Chang Hsun for Scarlet Fever. He himself developed high fever, sore throat, headache, swollen glands and later nephritis, etc. Within 36 hours, he was speechless and delirious, but with stimulants and serum he gradually recovered after 4 months. A younger son, seeing the serious condition of the father, remained in the same room for most of the time and even slept in the same bed. Three days after, he showed all signs of a severe infection with vomiting, pneumonia, etc. and died the same evening.

A married daughter (aet. 28), who had been nursing the father, also became ill and returned to her house with shivering, headache, sore throat, dry cough and severe vomiting. Her temperature rose to 106 with pulse of 126, when the rash appeared. Serum was given and after three days the disease abated and she slowly convalesced. Her three children, though isolated early, caught the infection, resulting in the death of the youngest.

- b.* Among the Chang Hsun family, which caused the above cases, one adult lady and two children died, though several persons were sick.

These rapid passages were duplicated throughout the Tientsin district during the winter of Jan.-Feb. 1923, and should give a serious warning to those in charge of public health work regarding the need of establishing more hospitals for the proper reception and care of such cases.

#### I. MANCHURIA.

From Manchuria, we have fuller statistics. The South Manchurian Railway has a well trained staff all along the line, where well-equipped hospitals are built. Their reports



therefore provide fruitful study. It is ascertained that Scarlet Fever was first seen in 1908. In 1909 two cases appeared in the Dairen area, then a few more along the line. Since then sporadic cases have been frequent, resulting now and then in small epidemics. From 1915 till Feb. 1924 Dr. Tsurumi was the Chief Medical Officer of the Sanitary Department, and to him we are obliged for the following information regarding conditions along the South Manchurian Railway.

In a report published in the Japanese Journal of Therapeutics (Feb. 11, 1922) Tsurumi dealt with two epidemics seen by him, namely, Jan.-July 1916, when 118 cases occurred, and Jan.-July 1920, when 137 cases occurred. The accompanying two tables are interesting:—

1911	101 cases with	22 deaths (21.7%)
1912	158 „ „	4 „ ( 2.5%)
1913	134 „ „	32 „ (23.8%)
1914	182 „ „	32 „ (17.6%)
1915	145 „ „	15 „ (10.3%)
1916	227 „ „	23 „ (10.0%)
1917	135 „ „	16 „ (11.8%)
1918	79 „ „	2 „ ( 2.5%)
1919	88 „ „	7 „ ( 7.9%)
<hr/>		<hr/>
1249		153 12.2%

*N.B.*—None of the patients older than 10 years. Sept. 1914–August 1916—163 cases admitted into Hospital. More than half under 5 years, others 6-10. More females than male. This corresponds to 1914 London statistics.

1916.		1920.	
Albuminuria	36, i.e. 25%	Albuminuria	18, i.e. 13.1%.
Lymphadenit.		Lymphadenit.	
cerv.	35, i.e. 24%	cerv.	47, i.e. 34.3%
Otitis media	21, i.e. 14.7%	Otitis media	10, i.e. 7.3%
Bronchitis	11, i.e. 7.7%	Bronchitis	23, i.e. 16.8%
Enteritis	8, i.e. 5.6%	Nephritis	15, i.e. 10.9%
Nephritis	2, i.e. 1.4%	Uraemia	1, i.e. 0.7%
Rhinitis	2, i.e. 1.4%	Haematuria	7, i.e. 5.1%
Urticaria	2, i.e. 1.4%	Others	16, i.e. 11.7%
Uraemia	1, i.e. 0.7%		

Tsurumi's conclusions are thus summarised:—

1. Out of 143 cases personally seen, all but three had tonsillitis and angina.

2. The symptoms were severe in 88, mild in 55. Enlarged tonsils predispose to severe form.
3. Vomiting and diarrhoea were seen in the early stages in one-third cases.
4. Lymphadenitis was frequent in 1920 epidemic. Out of 16 pus examinations, streptococci were seen in 13 (81.2%).
5. Anti-streptococcic serum produced good results. Out of 16 cases treated, 4 had immediate benefit (25%), 5 had fair benefit (37.5%), while the remainder showed little reaction.
6. Sero-vaccine was found satisfactory, especially in cases with severe angina.
7. Among 99 admissions into Dairen Hospital in 1920, 15 (15%) showed kidney complications. For three cases, saline infusion combined with sodium carbonate gave excellent results.

*Harbin.* The following figures are kindly supplied by Dr. Shapiro, in charge of the Harbin Town Infectious Diseases Hospital (mainly for Russians).

Year	Total admission Infectious Dis.	Scarlet Fever cases	Deaths
1920	377	22	1 (R)
1921	496	36	4 (R)
1922	739	63	5 (3R, 2C)

The only two Chinese admitted died; evidently they only went to Hospital after all hope had been given up at home.

A localised outbreak occurred in 1923-24 in the Refugee children's school organised by Madam d'Anjou (wife of the Commissioner of Customs). A short interval existed between two groups of cases, namely, the first lasting from Nov. 11 till Nov. 17, 1923 during which 4 out of 36 children were attacked; the second from Jan. 5th. till Jan. 19th, 1924 when 13 were attacked. The epidemic was mild and no deaths resulted. Several teachers and attendants complained of sore throat, but did not show any actual disease.

Our Medical Officers saw a considerable number of severe cases among Chinese in 1923. Among these was the family of a postal clerk (Fukien) who lost all his three children (aged 7,5,2) within the short space of 4 days (Nov. 25-28). Two cases were admitted into our Hospital on Dec. 8, 1923, received serum treatment and both recovered. These were apparently infected by neighbouring mild cases.



The son (aet. 8) of a prominent French resident suffered from a mild attack of scarlatina in 1922. Severe Nephritis and heart weakness later appeared and kept on for months, but patient recovered.

*Newchwang.* Dr. Phillips reported a considerable epidemic in 1918, when many foreign families were attacked with no deaths, as well as numerous Chinese cases with much fatality. Since then only rare and mild cases have been seen.

*Dalainor.* In October 1923, some excitement was caused by the sudden outbreak of Scarlatina in the coal mining village of Dalainor where pneumonic plague raged violently in 1921. Our Medical Officer Li An investigated this matter and reported as follows:—

*Scarlet Fever in Dalainor.*

Russion ..... 50 ,, (5 deaths) all male.  
1923-1924 Chinese ..... 1 case (no death)

Sex. 35 male, 16 female.

Age	1 year	.....	3	11 years	.....	2		
	2	„	.....	4	12	„	.....	0
	3	„	.....	5	13	„	.....	1
	4	„	.....	5	14	„	.....	1
	5	„	.....	9	15	„	.....	2
	6	„	.....	1	22	„	.....	1
	7	„	.....	3	30	„	.....	1
	8	„	.....	5	35	„	.....	1
	9	„	.....	5				
	10	„	.....	2				

Incidence month by month:

Oct. 1923 (19th) ..... 1 case  
November ..... 9 cases  
December ..... 7 ,,  
January 1924 ..... 30 ,,  
February ..... 4 ,, (last case Feb. 19.)

Evidently the epidemic was not very severe both among Chinese and Russians.

*Antung.* Scarlet Fever was noticed almost every year usually in a severe form. The Antung Medical Report for 1921 states: "In the autumn a severe epidemic occurred with severe mortality among children. In one family, all the children died within a few days."

*Taheiho.* This northern town of Manchuria was invaded by *Scarlatina* for the first time in October, 1923. Our Medical Officer W. H. Shih provides the following report:—

During the months of October and November I saw 22 cases altogether, including both Hospital and outside. All of them were children ranging from 2 to 10 years of age. Among the 22 cases there were five deaths and the other 17 cases ran a comparatively normal course. The following description roughly represents most of the non-fatal cases:

The onset is sudden with vomiting, headache and fever, followed later on by sore throat. On the following day the rash appears first on the chest and abdomen and later on extends to the limbs and buttock. The face is flushed and eyes are red. Pulse fast and full, goes up to 104 or 105. Urine scanty and cloudy. Bowels constipated. This generally continues till the end of 5 or 6 days when the fever subsides and gradually returns to normal with desquamation.

In one malignant case, a school girl of 8 years old, I noticed that the onset was nearly the same as the others, but more severe. On the following morning the rash came out abundantly all over the chest and abdomen also around the buttock. Within a few hours the papules were full of pus, so that to the naked eye they were white elevations instead of red. In the afternoon the patient was delirious and died on the third day.

As I was unable to obtain either anti-toxic serum or vaccine in *Taheiho* and *Blagoveschensk*, I treated the first few cases upon general principles, i.e., well-ventilated room, rest in bed, light diet, and painting of tonsils occasionally with formalin (1 in 200) and isolation.

Later on through the kindness of Dr. C. S. Lin I was able to obtain some anti-scarlatina vaccine from Harbin. I tried it on four cases, both of whom recovered, and also gave prophylactic injections to four children.

Of the five fatal cases, two were girls about 8 years old and died on the 3rd and 5th day respectively. The other three cases were a boy of 10 and two girls of 2 years old; all died of throat complications following suppuration of cervical glands.

#### M. GENERAL CONCLUSIONS.

1. *Scarlatina* is practically absent or very mild in South China, not unduly severe in Shanghai and Central Provinces, and severe in the North.



2. The disease seems to be comparatively more frequent among westerners residing in the country. In the north, although several deaths have been recorded among them, infection is usually not so severe as among the same classes of Chinese. The statistics of missionary families compiled by Dr. Lennox and briefly referred to elsewhere in this article form instructive reading.

3. The special virulence of the disease manifested when Chinese are attacked in epidemic form may be explained partly by its recent introduction into the country before any natural immunity is yet evolved. But the example of Japan, which owing to her complete hospitals and thorough system of notification and isolation has only allowed a mild and infrequent form to appear among the people, is sufficient to teach the responsible authorities in China the best and quickest way of subduing the disease.

4. Where septic conditions exist, treatment with a combination of anti-streptococcic serum and diphtheria antitoxin often produces satisfactory results. But in view of the rapid progress of the disease in individual patients, this method must be applied at the earliest possible opportunity.

5. From recent investigations upon the bacteriology of Scarlet Fever, especially by Drs. George and Gladys Dick, it seems that two substances of the nature of toxins play an essential part in the morbid phenomena of the disease, and that these toxins may be neutralised by antitoxins, thus bringing us nearer to a condition familiar in Diphtheria. Further, the injection of scarlet fever toxin may provide a method for distinguishing between susceptible and non-susceptible individuals in the same way that the Schick test has done in Diphtheria.

6. With the immense amount of material at hand in North China, especially Peking and Tientsin, it is hoped that much intensive research work may be carried out by trained men, of whom there are not a few now. Only by such means can the peculiar conditions in this region be satisfactorily studied with benefit to both foreigners and Chinese.

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(*Sen. Med. Off. Newchwang.*)

W. H. SHIH, M.D. (HONGKONG),  
(*Res. Med. Officer, Taheiho.*)

### COMPARATIVE STUDY OF SERODIAGNOSIS IN SYPHILIS.

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It is universally known that the Wassermann test in the serodiagnosis of syphilis holds a very important position in ascertaining the condition of a syphilitic patient who is undergoing treatment. But the procedure for the Wassermann test is so complicated that it cannot be relied upon unless the test is done in the hands of experts, as it has been stated by Boas.<sup>1</sup> "Die Serodignostik der Syphilis (Wassermann test) kann keineswegs jedem praktischen Arzt in die Haende gegeben werden. Es ist daher durchaus notwendig, die Untersuchung geeigneten Zentraldiagnosestationen zu ueberweisen." What Boas said is quite obvious; therefore, within the last few years there have been various attempts to simplify this test so as to make it handy for clinicians, but so far nobody has yet succeeded.

On the other hand, certain observers have worked along the line of precipitation. As this procedure is very much simplified, therefore many attempts have been made to replace the Wassermann test; e.g., Michaelis and Fornet-Schereschewsky ring tests, the lecithin reaction of Porges-Meier, Teruruchi-Toyoda's Kuorin test, Plausner's distilled water test, and Landau's Iodreaction, etc., which need not all be detailed here, are not at all universally applied.

Since 1917, Meinicke and Sachs-Georgi discovered the two precipitation tests, which possess the advantage of being very simple, and various authorities have confirmed the fact that its value is about the same as the Wassermann test. The theory of these tests has also been thoroughly investigated, and therefore it is expected that these tests will be universally adopted in the near future.

In Japan Kobayashi, Taoka, and Nishimura also discovered a ring test. The results, when compared with the Wassermann and Sachs-Georgi tests, have been very satisfactory.

When I was in Kitasato Institute, Japan, I made a comparative study of the Wassermann test, the Kobayashi-Taoka-Nishimura test.

The following technique was adopted with the results obtained therefrom:

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1. Boas. Die Wassermann'sche Reaktion, 1922.



## TECHNIQUE.

For details of these tests, one must refer to reference books; important points only are mentioned here.

*The Wassermann Test (Kitasato Institute Method)*<sup>1</sup>

*Antigen.* Take 0.5 c.c. of the ten times diluted cholesterinized alcoholic extract of guinea pig heart.

*Complement.* Take 0.5 c.c. fresh guinea pig's blood serum which has been previously diluted to 10-14 times according to the strength.

*Patient Serum.* Inactivated by 56° C.

Put 0.2 c.c. of the inactivated patient serum in the first tube and half of the amount of the first tube into the next tube, and so on until the sixth tube is reached. When the above three are well mixed add physiological saline to each tube in order to make up to 1.5 c.c. in each tube. Put into the incubator for one hour, then take out and add 0.5 c.c. of 5% sheep's corpuscles suspension and 0.5 c.c. of 800-1600 hæmolytic amboceptor. This is then put back again into the incubator. At the end of two hours it is taken out and the result read. Then place it in the ice chest and reread on the following morning.

*The Sachs-Georgi Test*<sup>2</sup>

The antigen for this test is well described in the original article.

The antigen I used in my experiments was made as follows:

Take alcoholic extract of bullock's heart and dilute it four times with alcohol. To 10.0 c.c. of the diluted extract add 0.45 of 1% cholesterin, but the strength should be such that its result must be identical with that of the Wassermann reaction.

Dilute the inactivated patient's serum ten times with physiological saline and put 1.0 c.c. of the diluted serum into a test tube used for precipitation. Then add 0.5 c.c. diluted antigen (1 c.c. antigen, 5 c.c. physiological saline).

- 
1. Takano-Kanai, *Ninsho-Jikken-Biseibutsu-Gaku*. Kobayashi, Chugwai-Iji-Shinpo, No. 951.
  2. Arbeiten a. d. Institut f. experimentallen Therapie u. d. Georg Speyer-Hause zu Frankfurt a.m. Heft, 10, 1920.  
Rubinstein, *Traité pratique de sérologie et de sérodiagnostic*, 1921.

Put into the incubator at 37° C. for one night, and read the result on the following morning. (Control is made according to the original method.)

*The Ring Test of Kobayashi-Taoka-Nishimura*<sup>2</sup>

The antigen<sup>3</sup> is prepared by adding 0.18 of 1% cholesterol to 1 c.c. alcoholic extract of bullock's heart. (The strength of the antigen should be such that its result must be identical with that of the Wassermann test.)

The percentage of various constituents in the antigen should be ascertained before the test. Before performing the test, the antigen is diluted ten times with physiological saline.

Put a certain amount of the inactivated patient's serum into an Uhlenhuth's test tube or a smaller test tube. Hold the tube slantwise and let the antigen flow in slowly along the wet place at the side of the tube, so that the serum and the antigen are on the two planes with the serum below the antigen. The technique for this test is identical with the Heller's test for albumen in urine.

When the instillation of the antigen is finished it is placed inside an incubator at 37° C. for two hours. Then take out and read the result. (It is advisable to put the tube at room temperature for ten minutes after it has been taken out of the incubator, as the result will come out better.) (Control is made according to the original method.)

Positive serum gives a white ring at the junction of the serum and antigen. The thickness of this indicates the servity of the case.

The following signs are used to indicate the servity of the serum :

- ‡ = Strong positive reaction.
- ⦿ = Positive reaction.
- + = Weak positive reaction.
- = Negative reaction.
- ± = Doubtful reaction.

*The Various Points to Be Observed in Performing Precipitation Tests.* Although the technique for the Ring and Sachs-Georgi tests is simple, still unreliable results have been obtained through negligence of minor points. Therefore, the technique and observations are equally important.

2. *Saikin-Gaku-Zashi*, May, 1921.

*Keio-Igaku-Zashi*, Vol. II, No. 2.

3. My own antigen, which also shows favorable results, will be published later on.



*The Various Points to Be Observed in Doing the Precipitation Test.*

1. The fresher the serum the better the result, as old serum undergoes certain changes which will affect the reaction. This is especially to be guarded against in the summer, as the bacterial growth is so easy and abundant.
2. Acid and alkali affect this reaction very much, therefore the various glass tubes employed should be clean and dry.
3. In performing the ring test, the column of antigen in the tube should be higher than that of the serum, otherwise not only will the result be made hard to read, but even the slightest shaking will mix them together.
4. In performing the ring test the tube should be shaken several times after patient's serum is put inside, so as to wet the wall of the tube and make the antigen flow down gently to avoid mixing.
5. In case the serum is blood stained, the ring test will be obscure and can only be made out by experts.
6. In diluting the antigens for the above two tests (Ring and Sachs-Georgi), the technique and amount of dilution will affect the result very much. This has been stated by various experts. Therefore it should be done according to the original method.
7. In performing the Sachs-Georgi test occasionally self-precipitated granules are met with, and so cause mistakes. The following points will serve to make a clear diagnosis:
  - a. In a positive reaction the color of the solution is lighter than that of the control. The granules are small and uniform, but in self-precipitated reactions the granules are not obtained by the interaction of serum and antigen, so the color of the solution is identical with that of the control. The granules are irregular in size and shape.
  - b. In case the result cannot be ascertained by the above method proceed as follows:

Take a typical positive tube as control and add NaOH solution to both tubes until the strength of the NaOH alkali grade reaches  $\frac{N}{100}$ . The true granules will disappear, while the false ones can only be dissolved, after adding a much stronger NaOH solution.

*Comparative Results of Wassermann, Sachs-Georgi and Ring Tests.* The following signs are used to indicate the result of these three tests: + = positive reaction, - = negative reaction, and  $\pm$  = doubtful reaction.

*A. Comparative Results of the Wassermann and Ring Tests.*

TABLE I

<i>Wassermann</i>	+	+	±	±	-	+	-	-
<i>Ring Test</i>	+	-	-	+	±	±	+	-
<i>No. of Sera</i>	156	4	5	8	10	5	12	343
<div style="border: 1px solid black; padding: 5px; margin: 5px auto; width: 80%;"> <div style="border: 1px solid black; padding: 2px; margin: 2px auto; width: 60%;">doubtful, 28</div> <div style="border: 1px solid black; padding: 2px; margin: 2px auto; width: 80%;">different result, 16</div> </div>								
similar result, 499								

TOTAL, 543 cases.

From Table I, we obtained from the Wassermann test and the ring test 92% with similar results, 3% with opposite results, and 5% with doubtful results. But those with doubtful results, either from the Wassermann test or the ring test, have to be reëxamined. Therefore, from the reëxamined doubtful cases adding up to 92%, we get 97% of similar result.

*B. Comparative Results of the Wassermann and Sachs-Georgi Tests.*

TABLE II

<i>Wassermann</i>	+	+	±	±	-	+	-	-
<i>Sachs-Georgi Test</i>	+	-	-	+	±	±	+	-
<i>No. of Sera</i>	150	17	3	10	10	3	15	337
<div style="border: 1px solid black; padding: 5px; margin: 5px auto; width: 80%;"> <div style="border: 1px solid black; padding: 2px; margin: 2px auto; width: 60%;">doubtful, 26</div> <div style="border: 1px solid black; padding: 2px; margin: 2px auto; width: 80%;">different result, 30</div> </div>								
similar result, 489								

TOTAL, 543 cases.

From Table II, we obtained from the Wassermann test and the Sachs-Georgi test 90% with similar result, 6% with opposite results, and 4% with doubtful results. Calculated according to the previous method, we get 94% of similar result.

*C. Comparative Result of the Sachs-Georgi and Ring Tests.*

TABLE III

<i>Sachs-Georgi Test</i>	+	+	±	±	-	+	-	-
<i>Ring Test</i>	+	-	-	+	±	±	+	-
<i>No. of Sera</i>	145	22	8	4	12	4	7	341
<div style="border: 1px solid black; padding: 5px; margin: 5px auto; width: 80%;"> <div style="border: 1px solid black; padding: 2px; margin: 2px auto; width: 60%;">doubtful, 28</div> <div style="border: 1px solid black; padding: 2px; margin: 2px auto; width: 80%;">different result, 29</div> </div>								
similar result, 486								

TOTAL, 543 cases.



From Table III, we obtained from the ring and the Sachs-Georgi test 90% with similar result, 5% with opposite result, and 5% doubtful. Total, 95% of similar result.

#### A SUMMARY OF THE RESULTS.

Now let us compare the above results. The ring test corresponds with the Wassermann test very closely, as there is only 3% of different results; even then, a certain amount of the sera used in the above tests are rather old and unfit for the ring test.

According to the report first issued by Kobayashi, Taoka, and Nishimura, out of seventeen hundred cases, 85% were identical with the Wassermann test. But later, and with the addition of the right amount of cholesterin, the result comes out very much better (please refer to their recent reports). Hayao and Kin made a thorough examination of the serum of dementia paralytica patients and the result obtained was 97% similar to the Wassermann test. Therefore, in taking the mean of the two, the result comes to 91%.

However, I also obtained excellent results on the Sachs-Georgi test, which nearly corresponds with the ring and Wassermann tests. Undoubtedly it is a good test. The results obtained by Georgi from Nathan, Mandelbaum, and Lipp and some other twenty odd experts, totally comes to 12,127 cases. The average comes to 92.44%. The results obtained by Kobayashi from Hubschmann, Stempe, and Messerschmid, etc., comes to 87.2%. The result of Kobayashi, Taoka, and Nishimura comes to 86%. The result of Taniguchi is 90%. From this we know that the similarity of the Sachs-Georgi and Wassermann tests comes to 85.90%, while my results came to 90%.

After referring to the reports of the above-mentioned observers and my own results, the identity of the three tests reaches above 90%. Therefore the two precipitation tests are not at all below the value of the Wassermann test; moreover, from my own experience it seems that the value of the Sachs-Georgi reaction is slightly below that of the ring test.

#### *Can Clinicians Apply the Precipitation Tests?*

The Wassermann reaction is so complicated with regard to its technique, materials, and control, etc., that it can only be done by specialists. Therefore it is impractical for clinicians. Though the value of the Ring and Sachs-Georgi tests is not identical with that of the Wassermann reaction, yet they are very simple; consequently, they are indispensable for clinicians in the serodiagnosis of syphilis.

As regards the choice of these two methods, it all depends upon one's inclination, but according to my experience I rather prefer the ring test, because:

1. It is easy to observe.
2. The procedure is simpler and can be done within a short time (two hours).
3. Comparatively the results are more reliable.

I am very much indebted to Professor S. Hata and Dr. K. Taoka for the valuable help they rendered me in making the above experiments possible.

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## SUMMARY OF PLAGUE EXPERIMENTS ON THE LICE OF TARABAGANS.

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It is proposed that a summary of the results of extensive plague experiments on the louse of the tarabagan be set down here. The detailed article has been sent to Germany and will be published in the scientific journals of that country.

Only brief statements and conclusions will be found in the following paragraphs, but because the data might throw some light on the whole plague and tarabagan problem, it was thought worth while to record them in these pages.

1. The louse of the tarabagan (*Linognathoides spec.*) can suck the blood of man, the ground squirrel and the guinea-pig, but will not live on a strange host for any length of time.

*4 experiments:* Ten lice, collected from a healthy tarabagan, were starved for 1-5 days and were put on the fore-arm of a man. A large percentage of them drew blood. The electric light did not disturb the act of biting which lasted half an hour. The bite of the lice did not cause much itching, but a female volunteer exposed to their bite said the sensation was disagreeable. It was observed that the lice often voided faeces in the act of biting.

Tarabagan lice, placed on healthy guinea-pigs, entirely disappeared after a few weeks.

2. When the host dies the louse of the tarabagan quickly passes on to another host.

*1 experiment:* Lice were put on a dying guinea-pig. After its death, the lice had all left and gone over to a living guinea-pig kept in the same cage.

3. Plague bacilli when sucked into the stomach of the tarabagan louse grow rapidly in clusters and finally fill up the lumen of the stomach.

*Observation:* Four hours after the ingestion of the plague infected blood, the growth of the bacilli could be observed. The accumulation of the plague bacilli clusters was to be seen

in the central part of the stomach where the blood was probably in stagnation, while in the periphery the same organisms grow in a diffuse manner, due probably to the disturbing peristalsis of the stomach wall.

Phagocytosis was seldom seen. The bacilli showed bipolar-staining and no involution forms were encountered.

4. Two or three days after the ingestion of plague blood, the tarabagan louse dies of infection, while healthy lice may live up to 10 days or longer when kept on a few hairs in test tubes and at room temperature.

*Experiment:* As the result of numerous experiments, one can state with certainty that all lice die after once ingesting plague-infected blood. The so-called self-cleaning was never observed. However, investigations with plague infected lice biting healthy or immunized tarabagans were not carried out.

5. The dead plague infected lice are distinguished by a dark red colour, and in transmitted light by a light red colour.

*Observation:* Microscopically, dark brown sediments of haemoglobin were seen in all parts of the body as well as in the intestinal tract. The trachea was of the same colour and was most distinct, so that the finest ramifications were clearly seen.

In the intestinal tract, clusters of virulent plague bacilli were found in pure culture.

No plague bacilli were ever discovered in other parts of the body.

6. The faeces of the lice contain plague bacilli in pure culture.

*Observation:* The bacilli were tightly packed in large clusters so that only at the periphery were bipolar-stained bacilli seen at all distinctly.

7. The alimentary canal of the healthy louse is nearly always sterile.

*Observation:* In the histological investigation, serial sections of over 100 partly healthy and partly plague-infected lice were made, but only on one occasion was a small lump of slender fusiform bacilli (40 microns in diameter) seen in the stomach of an infected louse.

8. Cocciform micro-organisms are found in the ovarian ducts of both healthy and plague-infected lice of the tara-



bagan, but never in the intestines. These micro-organisms are not pathogenic to guinea-pigs.

*2 experiments:* Pure cultures of these cocci were easily obtained as follows:—

8 lice, collected from healthy tarabagans, were washed successively with 10% acetic acid, 10% sodium carbonate, and thoroughly in six dishes of sterile normal saline. They were then ground up and the resulting juice was inoculated on agar. Guinea-pigs were infected with a large quantity of these germs in pure culture, but they did not fall sick.

9. After the death of the infected lice, the plague bacilli in the intestines do not show any involution forms until about the 5th day.

*Observation:* Before the 5th day, the plague bacilli were highly virulent, and it was only after that time that the virulence diminished and involution forms were observed.

10. Plague-infected lice collected from the dead tarabagan were seen to be infectious at least 13 days afterwards, though they were not protected from light or desiccation.

*Experiment:* The lice collected after the death of the plague infected animal were kept in Petri dishes. At fixed periods emulsions of 5-30 ground-up lice were injected either subcutaneously or intracutaneously into guinea-pigs:—

Time of injection after collecting the lice from dead host in days	Number of lice emulsified	Time of death of guinea-pigs in days	
		Subcutaneous	Intracutaneous
1	5	3	6
3	5	4	5
13	20	12	4½
25	30	alive	alive

11. By placing 40 lice collected 24 hours previously from a dead plague-infected tarabagan on a healthy sisel (*Spermophilus Eversmanni*, Brandt), one can infect it with plague.

*1 experiment:* The sisel succumbed to bubonic plague 150 hours after the lice were put on its neck. Plague bacilli were found in its bloody nasal mucus two days before death.

The faeces contained highly virulent plague bacilli.

12. Ten tarabagan lice, whose mouth organs are smeared with a virulent agar culture of plague bacilli, remained healthy.

13. Whilst probably all experiments of plague infected lice undergo changes described in paragraph 5, only one out of many experiments carried out with *B. anthracis* showed similar phenomena. In the alimentary canal of this louse, the anthrax bacilli were seen to have multiplied considerably.

14. The results of the observations outlined in Paras. 9 and 10 are in accord with those obtained by McCoy with the louse of *Citellus beecheyi*.

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*Serologist to the Service.*



## HOW I BUILT HOSPITALS IN CHINA.

WITH 5 PHOTOGRAPHS.

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I have often been asked to place on record an account of the method by which I have established the series of hospitals in various parts of the country, especially in view of troublous times through which China has passed during the last dozen years. Before doing this a few words of introduction may be necessary.

The year 1905 saw a great movement among all classes of Chinese to follow the progressive ways of the west. The Imperial authorities of Peking decided to adopt a Constitutional form of government and so despatched a Commission headed by Viceroy Tuan Fang with a staff of over fifty persons to different countries of the world to study the question. In Tientsin, Viceroy Yuan Shih Kai had accomplished splendid results in military, educational and commercial affairs through the assistance of keen, experienced, western-trained men, headed by Mr. Tong Shao Yi. The Imperial Army Medical College had just been started for the training of medical officers for his model army. I was then in the Straits Settlements having returned from England and Europe three years previously. Thanks to the recommendation of my old friend, Admiral Cheng Pi Kwang (who was later on—1917—assassinated by the agents of militarists in Canton), I was asked to come up to the north and assist in the organisation. I decided, however, to take a flying trip to England and Germany and study at first hand the methods adopted by the Royal Army Medical Corps in London and also by the General Staff in Berlin. Here I stayed for six months, and then returned to China in October 1908, only to learn that Viceroy Yuan Shih Kai had received Irish promotion to Peking as one of the Grand Councillors, and that both the Empress Dowager and the Emperor Kwang Hsu had recently died within a few hours of each other. Instead of my would-be patron Yuan Shih Kai, I found the powerful General Tieh Liang, his great rival (who died in 1920) installed at the War Office. Fortunately, his right-hand man was Admiral Tan Hsueh Heng, who as junior officer to my uncle, Captain Lin Kuo-chang, was in England during the years 1896-99 superintending the construction of Chinese warships at British shipyards. Through his efforts, I obtained an interview with General Tieh Liang and also my appointment as Vice-Director of the Imperial Army Medical

College. Whatever one may say against the dilatoriness or incompetence of Manchus, one has to confess that no finer or more courteous types of *gentlemen* existed in China, and such men as Tieh Liang, Yin Chang, Prince Chun (the Prince Regent, 1908-11), Hsi Liang (Viceroy of Manchuria, 1908-11), could not be equalled anywhere in the world for their kindness to strangers and juniors.

I assumed my new duties as Vice-Director of the Medical College, then situated at Tientsin, in November 1908, my immediated superior being Dr. Hsu Hua-ching. The teachers were mostly Japanese, who lectured in their language either direct or through interpreters. But the greatest handicap to success lay in the absence of a proper hospital, where the students could be taught clinical work. The only institution available was a small hospital situated in the centre of the city with a few beds, ill kept and poorly managed. Hence during my three years at Tientsin, I made frequent trips to Peking and interviewed successive Ministers and Vice-Ministers of War to plead for the establishment of a modern well-equipped hospital, where not only could students be efficiently trained, but soldiers could be properly cared for and the graduate medical officers continue their practice, so as to be ready for all emergencies.

But my words fell on deaf ears, and though plenty of money was found for uniforms and ammunition, none could be spared for a hospital. As a last resort, I asked for the construction of a model hospital to care for the twenty thousand odd Imperial troops stationed in the capital, but even this was not granted. One of the excuses was that our Chinese soldiers, like our Chinese people, did not care for western medicine, but my seniors forgot that in time of war native treatment would be of no use, and the few western-trained men in the army without regular practice could not cope with emergencies. Up to this day, in spite of revolutions and successive interprovincial strifes, no military hospital worthy of the name is as yet established in the capital and as a result mission hospitals or charitable hospitals like Dr. Gray's and the Union Medical College are frequented by men in uniform for ailments which should be treated by their own medical attendants.

It was not until the great Manchurian Plague of 1910-11 and its aftermath—the International Plague Conference held in Mukden under my Chairmanship in April 1911—that an opportunity came for the realisation of my ideals. Among the resolutions passed by that Conference of eleven nations, were the following :—



13. The need for isolation of pneumonic plague patients being urgent, permanent isolation hospitals should be available. Such isolation hospitals should admit of individual isolation, be of rat-proof construction, and be capable of easy disinfection.
42. A permanent sanitary nucleus should be formed, capable of rapid expansion in time of plague, and a list should be drawn up of medical officers who could be sent immediately to the affected area on the outbreak of plague.
44. With the view of giving effect to these recommendations, every effort should be made to organise a central public health department, more especially with regard to the management and notification of future outbreaks of infectious diseases.

At this point it may be necessary to classify the hospitals for whose construction I have been responsible into:

1. Those, where the initiative has come from me, e.g., Harbin and other Plague Prevention Hospitals.
2. Those initiated by local provincial authority; North Eastern (Mukden), Tsitsikar Hosp.
3. Those initiated by a non-governmental body and organised by me, e.g., Peking Central Hosp.

#### 1. *Plague Prevention Hospitals, Manchuria.*

The establishment in 1912 of the Manchurian Plague Prevention Service with headquarters at Harbin was a serious attempt made by the Chinese Government to give effect to the recommendations of the Mukden Conference mentioned above. Its inauguration was somewhat delayed by the Revolution, which started in October of the previous year, but fortunately the Viceroy of Manchuria (Chao Erh-sun), the Inspector-General of Customs (Sir Francis Aglen), the Vice-Minister of Foreign Affairs (Dr. W. W. Yen) and the late Commissioner of Customs (Mr. W. Haines Watson) all took a keen interest in the matter and did what they could to promote its success. For instance, the Viceroy appropriated from the Manchurian revenue Tls. 50,000. for the Hospital at Harbin, Tls. 40,000 for Manchouli, Tls. 30,000 for Tsitsikar and Tls. 20,000 for Lahasusu; Sir Francis Aglen induced the Diplomatic Body of Peking, which at first vetoed the scheme, to change their minds and to agree to the withdrawal of Tls. 60,000 annually from the Chinese Maritime Customs for the maintenance of the Service; Dr. W. W. Yen showed his sympathy by drawing up some of the regulations and placing

the Plague Prevention Service under the *aegis* of the Wai Chiao Pu (Foreign Office): lastly, Mr. W. Haines Watson (late Commissioner of Customs, Harbin, died 1914) who was present during the great plague of 1910-11 and therefore fully understood the urgent need of preventive measures, gave invaluable advice in the organisation of the several hospitals at the beginning of their existence.

Owing to the prevailing impression among western people that Chinese executives, however able or experienced, could not be trusted with money, it was agreed to appoint in addition to the Director and Chief Medical Officer, a Lay Director and Treasurer in the person of the Harbin Commissioner of Customs, "who will be responsible for the safe-keeping of the funds allotted for the maintenance of the Service, keep the accounts, issue salaries and supply the necessary funds for all expenditure duly sanctioned by the Director and Chief Medical Officer." On the other hand, in order to keep the Service efficient without undue interference from a layman, "the Director and Chief Medical Officer has general management of the Service, including the appointment, distribution and dismissal of the technical staff; he has also the power at his discretion to authorise appropriations to be made for any special purpose so long as that purpose is connected with plague and medical work and investigations, and provided the yearly limit of Tls. 60,000 is not exceeded." This division of labour has been found to work on the whole satisfactorily during the last twelve years, although one or two misunderstandings did arise through different interpretations of the regulations by succeeding Commissioners. A few words may now be devoted to each hospital.

(a) *Harbin.* The extensive plot of land, covering four English acres, on which the present Hospital stands, was once disputed territory claimed by the Russian Railway Administration. But, when we defined the object of our Service, General Horwath (then Head of the C. E. Railway Administration) gladly parted with it in 1911, and we now possess full title-deeds to this valuable property. The central office of our administration was at first situated in the Customs House, but as this arrangement was inconvenient to the Customs staff and ourselves alike, we moved to a rented building for two years, after which we occupied new quarters within our spacious hospital. Since the completion of our administration block in 1920, the office has been located here. The Harbin Hospital occupies two separate compounds, the west containing buildings devoted to administration and quarantine for 400 persons, and the east containing isolation blocks for the



accommodation of 30 suspects and 40 plague cases. This Hospital has been described in detail in the 1911-13 Report, and only the new additions need therefore be mentioned here. The original cost was \$70,000. Owing to the fact that the foundations of the several buildings were laid down towards the end of 1911, when the cold weather had more or less set in, it was soon found out that the walls showed a tendency to crack and the floors to rise in winter. Repairs were constantly necessary, and a few months after the World War was declared and when prices were rapidly rising, I suggested to the then Commissioner of Customs (Mr. Grevedon) to utilise some of the money we had saved during the past years in constructing a new double storey steam-heated hospital, but he demurred because of the uncertainties of the political situation. The opportunity passed, and before long the rouble (currency then prevailing throughout North Manchuria) rapidly declined in value until it reached practically zero-point, and as a consequence our hard-earned savings were totally lost. A fresh beginning was now made, and from October 1917 onwards our annual appropriations were paid in Chinese silver dollars instead of Russian currency. But both the staff and exchequer of the Service had suffered terribly. With the arrival of a new Commissioner of Customs (Mr. R. C. d'Anjou) in 1919, it was decided to build a new block, which would accommodate the plague research laboratory, museum, library and offices. In view of our limited funds, since no special appropriation was granted by the Government, the greatest economy had to be practised. We bought our own raw materials in winter and allowed no waste. The result was that we managed to complete a fairly large building having a wide foundation over seven feet deep with steam-heat and modern plumbing throughout for under Mex. \$18,000. This structure did yeoman service when Pneumonic Plague broke out in Harbin in January 1921, for, besides caring for 3125 plague patients (all fatal) from January to May, we were able to undertake extensive researches. These have been mostly described in the 1918-22 Report. One point, however, needs recording. On February 19th, 1921, Dr. Yuan Teh Mao, who had been on duty as Chief House-to-House Inspection Officer, showed symptoms of plague infection. I was at the time away at Suifenho (about 360 miles east) and returned to Harbin on the evening of the 20th. Owing to insufficient accommodation, Dr. Yuan and I had been occupying two adjoining rooms next to the Plague Laboratory on the first floor of the new building. His illness necessitated my removing to a poorly heated room in one of the barracks, where I was joined the next day (that is, the date of Dr. Yuan's death) by Dr. J. W. H. Chun, Dr. Pollitzer and Dr.



Young (a missionary colleague on a visit to us). Our cultures numbering some hundreds of tubes also occupied the same bedroom as ourselves during the disinfection of the new block, and we passed through some anxious nights. Dr. Yuan died early on Feb. 21st. This incident shows the danger to which our staff is exposed when insufficient funds are forthcoming for the accomodation of a medical staff who have to spend day and night within the precincts of a plague hospital in time of epidemic. Sixteen months previously (1919), when Cholera raged throughout Manchuria, we admitted 1962 seriously ill patients with only 275 (i.e. 14.11% deaths).

In 1922, another new block costing \$30,000 was added to the Harbin Hospital for the accomodation of general patients. This building contains two first class wards, two second class wards and four third class wards, totalling 45 beds. There is also a fine operating room as well as rooms for X Rays and photography. In May of this year (1924) foundations were dug for the erection of an up-to-date Laboratory for Plague, Serum and Diagnostic investigations, costing \$25,000. When this is completed we shall have separate new buildings for (a) Administration and Medical Officers Quarters, (b) General Hospital and (c) Laboratory, Library and Museum, and thus avoid further makeshift arrangements.

(b). *Sansing (Ilan)*. This is a town of about 15,000 inhabitants situated on the Sungari River, 150 miles north-east of Harbin. Owing to its being a port of call for river steamers, one of our hospitals was established there. For this purpose, some ready made buildings, numbering six large blocks within a fenced compound of  $\frac{3}{4}$  of an acre, were bought in 1913, and reconstructed so as to accomodate 60 persons. The cost approximated \$10,000.

(c). *Lahasusu (Tung Chiang)*. This small village of 1,000 people is situated at the junction of Rivers Sungari and Amur, and therefore occupies a strategic position. Although no plague had ever occurred here, it was considered necessary, in view of its importance as a port of call, to construct a modern isolation hospital in the neighborhood. Government land, measuring  $1\frac{1}{2}$  acres, was purchased at a nominal price, and new buildings costing \$20,000 were erected. These consist of a two storeyed brick building for the Medical Officer above and Out-patient Department below, as well as separate Quarantine and Observation Blocks. There is accomodation for 42 patients. During the troubles following the Russian Revolution, the "White" leader Kaminoff and his followers surrendered to the Chinese troops and were later on sent to Kirin. It was due to his attempt to escape from prison and to



obtain concealment in the local Russian Consulate that brought on the closure of all Russian consulates in China and terminated the recognition of the Russian Minister, Prince Koudaychoff, in Peking.

(d) *Taheiho (Helampo or Sakhalen)*. This is a growing city on the southern bank of River Amur and faces the Russian town of Blagovestchensk on the north. It is six days by steamer from Harbin. The Tsarist Government used to trace every infectious disease among man and beast, in Russian territory, e.g. anthrax, plague, swine fever, etc., to this locality, although it was, and is, one of the healthiest of places.

A fine brick hospital, functioning for normal and epidemic times, was built in 1913 and officially opened in 1914. It contains a large two storey block, as well as four quarantine and infectious blocks, accomodating 70 persons. The total cost was \$28,000. Our hospital has been the scene of many encounters between 'Whites' and Bolsheviks, both of whom applied for medical treatment and protection. The Japanese Military Red Cross also received our hospitality, and many Japanese wounded were treated within our walls during their fights with the Bolsheviks in 1920-21.

(e). *Manchouli (Lin Chung)*. This is the boundary town of Manchuria on the west adjoining Siberia. It has ever been the gate of entry for Pneumonic Plague from the endemic centres of Transbaikalia, and hence a hospital for observation and research at this place is absolutely necessary. In 1911 the Viceroy of Manchuria gave Tls. 40,000 for building a proper hospital at Manchouli. Foundations were forthwith dug in the spring of 1912, and much building material was purchased, but during the succeeding Revolution, the Mongols fanned by Russian expansionists burnt our place and left nothing behind but the bare ground. Several years then passed, and it was not until the second Manchurian Plague epidemic of 1921 came that we were able to borrow some buildings from the Municipality for our preventive work. Since that time we have stationed a permanent sanitary staff at Manchouli, sending expeditions into Siberia whenever occasion demands it and cooperating with our Russian colleagues in the happiest way. In 1923, we bought a ready built stone house for \$9,000 for the purposes of a research laboratory and quarters for our medical officer, at the same time retaining a large wooden block lent by the Municipality as Polyclinic. At this station is situated the large apparatus for disinfecting tarabagan skins with formalin gas before export. At present there is an embargo upon export and hunting by the Heilungkiang Governor, otherwise millions of skins could be disinfected and exported with benefit to all concerned.

(f). *Newchwang.* The Quarantine Hospital of Newchwang is the latest addition to the Manchurian Plague Prevention Service. For several years the need of such a hospital had been realised by all classes of the community because of constant invasions of Cholera from Shanghai, Japan and Korea. But it was not until 1918 that the appropriation was finally sanctioned. Building operations were commenced in 1919, and the hospital was formally opened on July 10th, 1920. There is a large front block 162 feet wide containing operation and diagnostic rooms and general wards; next is a disinfection block and behind lies the contagious block with individual rooms and verandahs facing the south. The cost of these original buildings is Tls. 40,000 and accommodation is for 45 beds. In 1923 a series of six detention blocks, built of bricks and having cement floors, were added to the hospital. Each block possesses a set of hygienic *kangs*, insect-proof and dirt-proof, to serve as beds for those detained under observation. There is accommodation for 80 persons in each block, and hence a minimum of 400 persons may be detained at any one time. The cost of this second lot of buildings is \$30,000. The total ground covers  $3\frac{1}{2}$  acres and lies on the south bank of the River Liao.

2. *Those initiated by local provincial authority.*

(g). *North-Eastern Hospital, Mukden.* One of the results of the internecine war between General Chang Tso Lin on one hand and General Wu Pei Fu on the other in 1922 was the awakening of the military leaders to the need of providing qualified surgeons for the hundreds, nay thousands, of soldiers wounded by modern fighting weapons. Both sides at the time had to rely mainly upon foreign doctors and institutions for the care of their wounded. Hence, in addition to the employment of younger, energetic Chinese to replace the older opium-sodden officers, General Chang and his son decided to construct a model military hospital in Mukden, partly to treat their soldiers and partly to train medical officers. For this purpose they entrusted me with its planning and organisation. A brilliant Chinese architect, S. S. Kwan, trained in America, was employed to draw the plans, the condition being that the hospital could be used, whenever necessary, for the public as well as military. The verandah system was chosen, with a central two-storey block for administration and officers ward, and individual unit wards radiating from a central pathway. Each ward possesses a complete heating and plumbing plant as well as rooms for nurse, clinical diagnosis, service, and lavatories. Altogether 26 out of 35 blocks have so far been built. Besides wards, there are three operating rooms, all



equipped in the most up-to-date manner, separate departments for venereal diseases, skin, otolaryngology, eyes, and accidents. A deep artesian well has been dug, from which pure water is pumped with an engine to a high tower. There are platforms to which the railway track loads. The total cost is over \$600,000. including \$140,000. for plumbing and heating. There is accomodation for 400 beds. This North-Eastern Hospital is easily the model military hospital in China, although many defects are still present. It is hoped that an efficient medical staff may be employed to run the hospital which will be opened this summer (1924). See photo.

(h). *Tsitsikar (Pukuei)*. This city, the capital of Heilungkiang Province, was given Tls. 30,000. by the Viceroy of Manchuria for the establishment of an anti-plague hospital in 1911 as a result of the great epidemic. The local Taoyin, Sung-Siao-lien (later on promoted to Governor of the Province) requested me to help in planning and equipping the Hospital, which was formally opened in 1912. There is accomodation for 60 patients. The Medical Officers are not directly appointed by me, though my assistance has been frequently sought in times of epidemic. The running expenses are provided by the local authorities.

3. *Those initiated by a non-governmental body and organised by me.*

(i). *Peking Central Hospital*. This is the institution to which I devoted my best efforts continuously for four years, because it was intended to make it the model *civil* hospital of China. The scheme originated with a private visit made to Mr. Chou Hsueh Hsi, (Minister of Finance during President Yuan Shih Kai's term in 1915) who was desirous of establishing a sanatorium costing \$100,000. at the Western Hills, where he used to spend regular week-ends. I pointed out to him the more urgent need of an up-to-date general hospital in the capital, which could serve as a model for both officials and people, and thus promote the interests of scientific medicine. The sanatorium could then be built on a smaller scale as an adjunct for convalescents. The upshot was a meeting of influential Peking residents in the spring of 1915 at the Central Park Committee Room. Among those present were Messrs. Chou Hsueh Hsi (Minister of Finance), Chu Chi Chien (Minister of Interior), Tsao Ju Lin (Minister of Foreign Affairs), Chang Chung Hsiang (Minister of Justice), Lin Chang Min (Chief Secretary of the Cabinet), C. C. Wang (Councillor, Ministry of Communications), Sze Sao Tseng (Director-General, Lung Hai Railways), Chief of Police Wu, myself and a dozen others. \$110,000. were promised at the meeting besides the \$100,000

at the disposal of Mr. Chou. A fine plot of government land in the west city next to the historical Temple of Imperial Ancestors was presented to the Hospital, and another acre situated behind was afterwards bought for \$13,000. to complete the site. An American firm of architects was engaged to draw up suitable plans, the building contract was given to a German, and I was appointed Honorary Medical Director to supervise everything except the finance. Foundations were dug in June 1916. The uncertainties all over the country brought about by the Japanese 21 Demands and Yuan Shih Kai's attempt to become Emperor considerably hindered the campaign to procure funds. This meant more intensive efforts on the part of the organisers, and greater need for economy in every direction. For instance, the Government Railways charged half rates for all materials conveyed by them; the Maritime Customs and Peking Octroi allowed free import to our goods; steel rods for the concrete work were supplied by the Hanyang Ironworks at 20 percent discount; the Chee Hsin Cement Co. deducted 15 percent from their regular prices; Mr. Sze placed his accountants at our disposal, and neither Treasurer nor Medical Director received any salary. Even the British firm of Twyford & Co., (with Mr. Thomas at the head) offered their services as our Tientsin agents without any commission. Everywhere I went I solicited subscriptions and travelled as far as the South Seas to interest wealthy friends who subscribed \$30,000. I myself donated \$2,500. Through a personal appeal to Mr. Liang Chi Chiao (Minister of Finance 1916), he granted a further \$30,000 from the Ministry as well as an annual allowance of \$1,000. for upkeep. Valuable medical and surgical instruments were also presented by Admiral Sah Chen Ping, then in charge of the Arsenal at Tehchow. Thanks to such hearty co-operation from all sides we were able to build and equip a thoroughly modern ferro-concrete \$400,000 hospital for only \$300,000. There is accommodation for 10 first class, 20 second class and 120 third class patients. A full account of this hospital appeared in the *Modern Hospital* (America), April 1917.

The new Hospital was formally opened on Jan. 27th, 1918, the whole of Peking turning up for three days to inspect it. But an unfortunate dissension soon occurred between the Treasurer, Mr. Sze, and myself. We had been working harmoniously for three years during the formative period, and no person could have been more liberal or keener on the success of the institution. At the completion of the building, however, he was advised by interested persons to create for himself the post of Hospital-lord (院董) with power to control the actions of the Medical Director instead of cooperating with him



as an equal, as he had hitherto done. Many friends intervened to mend the rift, but there was already another doctor waiting to replace me on my departure. I resigned a short time after the opening, and was thus prevented from realising my ambition to *run* as well as *build* the hospital as a model institution, where our Chinese medical men and women, rapidly increasing in numbers, could find a joint meeting place for their professional activities. Nearly six years have now passed since its establishment, and this great hospital, to which so many friends devoted their time and money, is being managed, as so many government institutions in Peking are, without vigour, enthusiasm or efficiency, although ample funds have been provided for maintenance and improvement.

*Other Activities.* Besides the above permanent hospitals, I was successful in inducing the Central Government in 1911 to build a series of quarantine sheds at Mukden, Koupangtzu, Shanhaikuan, and Newchwang as precautionary measures against the plague. These were all erected in haste during the winter months, of corrugated iron sheetings without wooden floors. The cost of each varied from \$20,000 to \$40,000 according to capacity. The minimum accomodation was 1,000, and the maximum 3,000. All these have now tumbled or been torn down except those at Newchwang and Shanhaikuan. When the Rockefeller Medical Mission visited China in 1915, I was invited by Drs. Welch, Flexner and Peabody and Mr. Roger Greene of the Commission to express my opinions as to the advisability of establishing a Medical College and Hospital in Peking and also to the kind of language in which medicine should be taught. The unrivalled Peking Union Medical College and Hospital, opened in 1921, are now included among the sights of the capital. In 1917, I petitioned the Foreign Office and Ministry of Finance regarding the need of establishing a Central Hygienic Laboratory in Peking with which our Manchurian Plague Prevention Service could be affiliated. The dispatch sanctioning my petition with the necessary appropriation had actually been received at the Foreign Office from the Ministry of Finance, but the next day it was withdrawn. Fortunately, after the outbreak of Pneumonic Plague in Shansi (1917-18) the surplus left from the one million dollar loan from international bankers was utilised for the building and equipment of this Hygienic Laboratory in the historic grounds of the Temple of Heaven. This Institute is now turning out useful vaccines and sera and receiving an annual grant of \$110,000. from the Maritime Customs. There have never been more numerous or more capable Chinese medical graduates in China than at this present moment, and it is hoped that every one will regard it as his bounden duty to push forward the

interests of his humane profession for the promotion of public health as well as the mere routine treatment of the sick. Apart from public hospitals, private ones are essential, but these should be properly equipped with up-to-date instruments for diagnosis and treatment, not necessarily in a lavish or wasteful manner. Only by this means can Chinese hope to win their conservative people over to a just appreciation of the benefits of modern medicine.

## SUMMARY OF PERMANENT HOSPITALS ESTABLISHED.

<i>No.</i>	<i>Location</i>	<i>Year</i>	<i>Accommodation.</i>
1 a	Harbin (i) .....	1912	470.
	„ (ii) .....	1920	Administ. and Lab.
	„ (iii) .....	1922	Gen. H. 45.
	„ (vi) .....	1924	Lab. and Museum.
b	Sansing .....	1913	60.
c	Lahasusu .....	1912	42.
d	Taheiho .....	1913	70.
e	Manchouli (i) .....	1921	30.
	„ (ii) .....	1923	Lab.
f	Newchwang (i) .....	1920	Isol. and Gen. 45.
	„ (ii) .....	1923	Quar. 400.
2 g	Mukden (N. Eastern) ...	1924	Gen. and Milit. 400.
h	Tsitsikar .....	1912	Gen 60.
3 i	Peking (Central) .....	1918	Gen. 150.

WU LIEN-TEH.





Two new buildings of Harbin Hospital. In front is the Office and present laboratory, completed in 1920; behind is the new General Hospital completed in 1922.

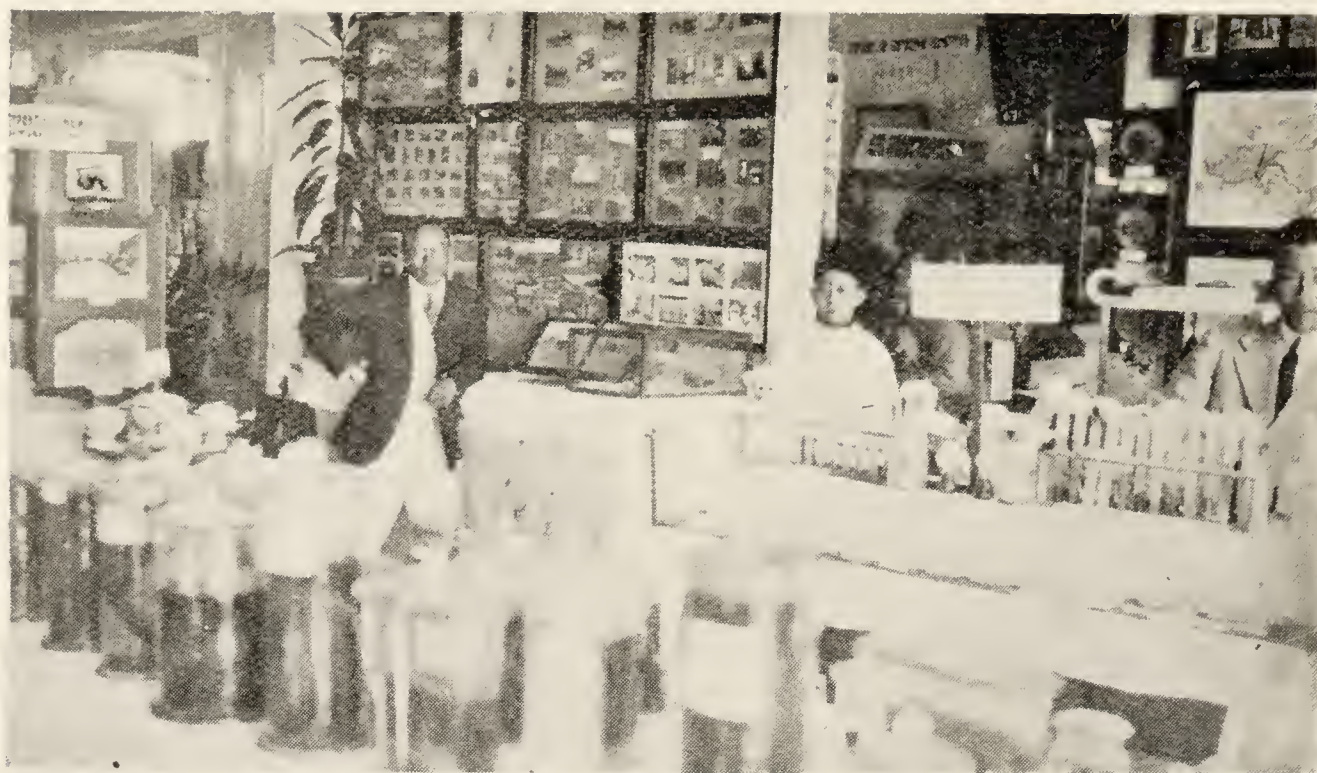
者後成落九民於室驗化現者前居樓新兩之院哈  
成落二十民於院醫新



One of the medical wards in the new Hospital.

部一室病養科內內院醫新





Exhibition of the Plague Prevention Service during 25th anniversary celebration of Chinese Eastern Ry., 1923.

圖列陳處總疫防省東會覽博念紀週五十二路東中二十民



Tarabashine or Siberian eagle feeding upon tarabagans.

亞利比西或鳥獺嗜  
圖獺旱捕食鷹



Laboratory of Service at Manchouli, permanently established in 1923.

室驗化里洲滿之成落二十民



AS A NATIONAL GUEST IN JAPAN.

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Early in August, while on my way from Harbin to Peking, to consult the Government about the Far Eastern Conference of Tropical Medicine to be held at Singapore in September 1923, I was verbally invited by the Japanese Consul Nishi at Changchun to visit Japan as a national guest and deliver a series of lectures at the principal universities. This I consented to do as soon as the necessary permission was obtained from our Foreign Minister. After this I proceeded to Singapore, attended the Conference there, September 3-17 and returned to Shanghai on October 6th. There the Japanese Consul General Yada requested me to take the first steamer for Nagasaki, where a celebration was to be held on the 10th in honour of the hundredth anniversary of the landing of the Dutch doctor Siebold who was the first European physician to teach Japan western medicine. Hence I left that same evening on board the "Empress of Russia," after having returned only a few hours previously from the south.

On arrival at Nagasaki a large group of Japanese doctors headed by Dr. Yamada (formerly Director of the South Manchuria Medical College, Mukden, now Head of the Nagasaki Medical College) and including Prof. Shimidzu and other members of the staff, Dr. Asagi (Chief Municipal Doctor), Dr. Yamada (Harbour Master), Tsuruda (Port Health Doctor), Dr. Todyo from Kagoshima and Amenamori (Doyen of the Medical Faculty), met me on board and conducted me to the Nagasaki Hotel. Owing to the recent earthquake at Tokio and vicinity, unnecessary formalities were done away with, but the Japanese friends did their utmost to welcome me. For the same reason the Siebold anniversary was postponed and I spent the next few days in sight-seeing besides taking a share at the Chinese Consulate in the Republican anniversary and inauguration of the new President Tsao Kun. One of the most interesting sights at Nagasaki was the Quarantine Hospital, the first of its kind in Japan, and claiming as its former medical officers such distinguished names as Dr. Takaki and Dr. Nagayo (father of the present Professor of Pathology at Tokio).

On the morning of the 11th I left by train for Kagoshima, a delightful spot on the southernmost part of Kyushiu Province facing the volcanic island Sakurajima which erupted fiercely ten years ago. The journey of 280 miles took 13½

hours. In this city I begged to be allowed to stay in a native hotel and was therefore conducted to the *Satsumaya*, thus living in truly Japanese style. We took a steam launch to inspect the volcanic island where plenty of lava and brimstone had been shot out from the crater, thrown into the harbour and had thus narrowed the channel by one third: Early at 8 a.m. the next day the Kyushu Conference was opened and attended by over 800 physicians from all over the country. Prof. Sata, President of the Osaka Medical College, delivered the first address on the latest investigations upon tuberculosis, of which he had made a special study in Germany and since his return. This address took one and a half hours. I was the next speaker, and after some preliminary remarks, dwelt lengthily in English upon the role which the tarabagan and other wild rodents played in the epidemiology of plague. Dr. Todyo kindly interpreted the essence of my speech from English into Japanese. The whole of next day (Sunday) was also devoted to the Conference and over 60 papers were read or accepted. To me it was remarkable to see the number of papers presented at this and other Japanese Medical Conferences; every member seemed to regard it as a duty to contribute something, and even when owing to lack of time this was not read, the rebuff was taken in a most cheerful spirit.

Kagoshima was famous for the stand which the early *daimios* and *samurais* made in upholding their lost cause before the Restoration and the thousands of little mounds on the top of the hill bear witness to the courage with which they fought and died.

I left Kagoshima on the evening of the 15th and reached Fukuoko (capital of the Province and seat of Imperial University). The Head of the University was Dr. Mano, a distinguished engineer, who had studied at Glasgow and therefore spoke English perfectly. In the city I met also my old friend Kubo, perhaps the greatest specialist on otolaryngology in Asia. Here we spent an interesting day, visiting clinics as well as the Temple of Shigahara, whom the Japanese regard as their Confucius.

After travelling all night, Prof. Sata and I arrived at Osaka next morning. This is indeed a big city and since the fire at Tokio has become the biggest in the land. I went round the new City Hospital, designed and promoted by Dr. Sata. This is the first ferro-concrete hospital building in Japan. The land has been given by the Municipality, whilst the cost of construction has been met by subscriptions from the leading citizens including a banker, who donated one million yen. The total cost is four million yen. In order



to make every possible use of the building, the polyclinic departments are placed on the ground floor, the operation rooms and laboratories on the first floor, large wards on the second floor, and various lecture rooms on the topmost. Adjoining this building is another ferro-concrete structure, completed two years ago, containing mostly in-patient wards. There is accommodation for 400 patients altogether.

Dr. Sata is certainly a great organiser for nowhere in Japan have the general public contributed such large sums for the erection of a hospital. I regret to learn that since my return to China he has vacated his important posts as President of the Medical College and Head of the Hospital.

On the 19th I visited Kyoto and gave an address in the University Anatomy Lecture Hall on "Practical Hygiene in the Orient." President Araki of the University presided at the meeting supported by Prof. Adachi (Anatomy), Hirai (Pediatrics) and Akira Fujinami (Pathology). Altogether 460 members of the staff and students attended. At the special request of the staff I spoke in the mandarin dialect which was translated into Japanese by a Chinese graduate, Dr. Wen.

On the 20th I delivered an address on "Researches upon Plague in China" in the large Conference Hall of the Osaka Medical College. This lecture was delivered in English and translated by the Professor of Pathology.

The ancient capital of Kyoto interested me particularly because of its old associations with China at the time of the Tang dynasty. Here one could see pictures, buildings, customs, dresses and ceremonies similar to those recorded 1200 years ago. Even the resorts of pleasure took pride in displaying the pictures and utensils handed down uninterruptedly since the Ming period. In the religious town of Nara are manifested many peculiar emblems of an ancient and almost forgotten past. The oldest Buddhist Temple in Japan is found at Horyuji, 7 miles from Nara, where priceless treasures, 1300 years old, are housed in unbroken array.

Besides the new City Hospital at Osaka the authorities have built an up-to-date Public Health Laboratory with Dr. Fujiwara as the Head. Here men and women were busy preparing models and pamphlets for public demonstrations, according to the latest American methods, besides performing the usual routine work of analysis, statistics, etc.

Though the Japanese have a tendency to laugh at Kyoto for its conservatism in the same way that Englishmen laugh at Oxford, the Professor of Hygiene (Dr. Toda) is certainly

a very live man. He is not satisfied, as so many Japanese hygienists are, with being merely advanced bacteriologists, but takes his stand as the acknowledged leader of a group of young go-ahead doctors trying their best to apply the resources of *all* medical sciences for the prevention of sickness and suffering. With this object in view his researches and experiments often bear a practical aspect, especially applicable to the needs of his surroundings. If all health problems in the East are tackled in the same way, I am confident that tuberculosis, scarlet fever, intestinal diseases, goitre, leprosy, etc., will be easily controlled within a short time.

At Kyoto I was joined by Dr. Tamiya (Assistant to Prof. Nagayo), who had been sent from Tokio to escort me to the burnt capital. As the through trains between Osaka and Tokio were not then running, we decided to take the steamer from Kobe to Yokohama. The two fast steamers "Nagasaki Maru" and "Shanghai Maru," running between Shanghai and Nagasaki for the last two years, had been commandeered by the Government soon after the earthquake to ply between these two ports.

So on Oct. 22 we left Kobe at 10 o'clock in the morning, engaging the Imperial suite on the "Shanghai Maru." Early the next day we arrived in Yokohama and witnessed an extraordinary sight. Many ships were in harbour but practically all the wharves and godowns had been burnt, destroyed or twisted. Half of the break-water had actually sunk beneath the sea and just left a line on the surface of the water to show its presence. On land everything appeared to be in ruins. A few walls, such as those of the Yokohama Specie Bank, seemed intact, but within all had been gutted. The once beautiful British Cemetery was all torn up and the marble tombstones laid flat. The Grand Hotel was represented by a mere chimney and under the debris had been buried 300 guests, who had assembled at noon of that fateful day as usual to discuss the day's news and latest share quotations.

We motored from Yokohama to Tokio on 23rd instead of taking the train. About 20 villages were passed on the way, few of which seemed to have suffered much. Apparently the fires were not severe there. We approached Tokio through the Shinagawa district. At least 5 of the 16 districts had been completely levelled to the ground. The famous Ginza (Oxford Street of Tokio), Nihonbashi (corresponding to Wall Street of New York), Imperial Theatre (modelled after the Paris Opera house), Okura's Museum (recently presented to the Government), Asakusa (Amusement quarters, including the



Yoshiwara), Municipal Hospital, International Hospital (Dr. Teusler's) and other notable buildings too numerous to mention, were no more. Strange to say, the two leading hotels, the Imperial and the Tokio Station, had been left intact. The former was a strange building, designed by the American artist Wright, who combined ferro-concrete stability with oriental artistry and cost the owners seven million yen. The Tokio Station Hotel was a red building, modelled after the Hamburg station and, though not built of concrete, had withstood all the shocks and subsequent fires. Rooms had been assigned to me at the Imperial Hotel by the Japanese Foreign Office. That same afternoon I called upon the Minister Baron Ijuin (whom I had known as Japanese Minister in Peking in 1911 but I regret to say died on April 25, 1924), Vice-Minister Matsudaira (formerly Consul General in Tientsin) and Mr. Debuchi (Chief of the Asiatic Affairs Bureau).

Next day I visited the University and saw the great havoc which the fire had caused. The unrivalled Library, the Biochemical Laboratory, the Science Building (including Botany, Zoology, Chemistry), Physiology, Pharmacology and Technology Laboratories, had all disappeared. I was informed that when the first quake appeared, some bottles containing inflammable stuff fell down, spontaneous combustion took place and fire first resulted in the Chemical building, from which it spread to other quarters. Fortunately the hospital wards were only slightly damaged and enabled the doctors to admit hundreds of urgent cases. I called upon Professors Kitasato, Nagayo, Irisawa, and Miura. On the 25th I visited and saw Drs. Hata, Kitajima, Miyajima, Miyagawa, Ishiwara, Kanai and others. I saw also the Municipal Red Cross Hospital with Dr. Tamura (formerly of Tientsin) in charge. Over 600 refugees, mostly women and old men, were housed in the former Exhibition Palace at Ueno Park. Everything was simple, but neat, and the fortitude of the sick and homeless was wonderful.

That same evening a grand banquet was given in my honour by the Foreign Office and presided by Vice-President Matsudaira. Cordial toasts were exchanged and I urged my Japanese friends to treat our Chinese students as they would theirs, passing the capable and rejecting the incapable. In that way only could useful men be produced for the mutual benefit of the two countries.

On October 26 I gave my lecture at the Imperial University on "The Interdependence of Japanese and Chinese Medicine." This lecture is published in the present Report.

The whole medical staff and students attended. Dr. Miyagawa translated my English sentences one by one into Japanese. That same evening the Tokio Medical Association, the most powerful in Japan, entertained me to dinner at the Seiyoken Restaurant with Professor Kitasato in the Chair. Besides the Chairman, there were present such distinguished doctors as: K. Ishiwara, M. Miyajima, R. Inada, K. Kusama, T. Nakahara, S. Uchiya, Y. Yamaya, R. Sado, T. Tamija, J. Uchida, M. Incuye, W. Okada, K. Suzuki, Ch. Yokote, H. E. Kanasugi, T. Kitashima, S. Hata, T. Irisawa, K. Miura, T. Sato, H. Shihara, M. Nagayo, H. Yamada, S. Kinoshite, H. Iwata, Y. Miyagawa, I. Takaki, Z. Hatta, M. Segawa, M. Fukushi, S. Kansi, M. Kanno, Kingo Goto, Kozo Saisawa. Very cordial speeches were again made by Dr. Kitasato and myself. I urged that an Institute for Experimental Science might soon be established in China from the funds of the returned Boxer Indemnity, where the best Japanese and Chinese scientists might work together for the benefit of mankind.

I left Yokohama on Oct. 30 by the T.K.K. "Shinyo Maru" and returned to Shanghai on Nov. 5. Before my departure from Tokio I submitted to certain friends a scheme for the utilisation of the Indemnity funds for promoting cultural relations between Japan and China. The following is a summary:

#### SCHEME FOR THE PROMOTION OF CULTURAL RELATIONS BETWEEN JAPAN AND CHINA.

It is estimated that about 2 million yen are available from the Boxer Fund for an indefinite period.

I suggest that one half of this be utilised for scientific and medical purposes. Reasons:—

- a. Scientific education makes for progress among the people. China is backward, because her leaders and educators are backward in science. Science makes for better understanding.
- b. Americans and Britishers have established several colleges and hospitals for benefit of Chinese. Japan has done little in this respect, and this is interpreted in an unfavorable light by others.
- c. Promotion of preventive medicine in China makes for greater security in Japan during epidemics, and hence less outlay in her own preventive work.



- d. Substantial assistance towards the prevention and alleviation of suffering is the strongest and surest way of winning everlasting friendship.

### Methods of application.

1. A Library in Peking, containing modern practical scientific as well as ancient literary books of all nations, and similar periodicals.
2. An economically established but absolutely up-to-date medical college and hospital in Canton City—the prime mover of all progress in China, and the great centre of modern thought. Roughly speaking, this combined institution will cost:—

Land perhaps presented by the provincial government.

College buildings and equipment.....	\$900,000	spreading over 3 years
Hospital       "       "       "       "	\$300,000	"       "       "       "
Annual expenditure of both .....	\$450,000	"       "       "       "

The Hospital will derive considerable income from fees of patients as the Cantonese dislike free treatment.

3. A popular Museum of Practical Hygiene in Peking for the teaching of health preservation on the model of the Institute started by Whitewright in Tsinan-fu. In this Museum will be displayed all matters pertaining to body constitution, food and clothes used in different parts of China, analysis of Chinese medicines and methods of treatment, causes of diseases in the East and their prevention and treatment, health inspection in schools, home hygiene, infectious diseases and quarantine. Everything to be placed upon a scientific, yet popular and practical footing, so that all, including officials, scholars, students and masses may see and understand. Lectures, pamphlets, movies, etc., to be included.

Cost of building .....	\$300,000	Many will be presented and made specially by local men under proper supervision.
„ displays .....	\$150,000	
Land .....		
Annual Budget .....	\$ 60,000,	will probably be presented by the government or Manchu House, to be increased later.

This Museum may be situated next door to Library and even affiliated with it.

4. An Institute for Experimental Medicine and Science in the Yangtse Valley, preferably near Shanghai or Nanking.

Here diseases may be investigated and research made into scientific problems affecting both China and Japan for their mutual benefit. Extraordinarily interesting scientific problems affecting the happiness of mankind still await solution, and oriental savants ought to build up a reputation for themselves by putting their heads together instead of working separately as they have hitherto done. The surest way of showing Asia and Europe that the East has a culture and original mind of its own lies in this mutual cooperation in scientific investigation. The best brains of Japan and China should be employed in initiating this Institute, and the time will soon come when great geniuses, now largely wasted, will be attracted to this scientific Mecca, and thus re-establish the name of the East as the original home of culture and science.

Estimated cost of building...	\$600,000	spread over 3 years
"      "      " equipment	\$300,000	"      "      "      "
"      "      " land.....	\$200,000	if in Shanghai
		(if in Nanking, presented by gov.)
Annual budget .....	\$200,000	for first three years.

WU LIEN-TEH.



## THE INTER-DEPENDENCE OF JAPANESE AND CHINESE MEDICINE.

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*(Abstract of a Lecture delivered at the Imperial University  
of Tokyo, Oct. 26, 1923.)*

Permit me at the outset to express my high appreciation of this invitation to lecture at the Tokio Imperial University, the most famous of all Japanese Universities. This is an honour not only to me but to my Chinese colleagues and Chinese medicine.

Secondly, let me express the heartfelt sympathy of the Chinese medical world with the Japanese nation in the widespread suffering caused by the earthquake. Particularly are we sorry to notice the losses entailed by education and science in the ruin of so many famous scholastic institutions, including the University, where the Library, Technological, Physiological, Bio-Chemical, Science and Pharmacological Institutes are no more. At the same time, the wonderful bearing and courage of the people, from the highest to the lowest, in the face of unparalleled adversities, and the rapidity with which normalcy is being regained, are a lesson to the world in general.

In passing to the subject of my lecture to-day, I fear that those who have honored me by attending will be somewhat disappointed at my choosing such a commonplace matter to talk upon. At Kagoshima, I lectured upon "The Latest Investigations upon the Tarabagan" which had yielded valuable results during the last two years. At Kyoto, I dealt with "Certain aspects in Practical Hygiene of the Orient." At Osaka, I spoke upon "Researches upon Plague in China" which as you know is my special subject.

Hence, in order not to repeat myself, particularly as this will be the last opportunity I shall speak at a University in Japan, I thought the subject of "Inter-dependence of Japanese and Chinese Medicine" might be suitable.

Many of us have no doubt thought upon the subject and have probably come to the same conclusions.

Our race and our culture are the same. Up to sixty years ago, Japanese culture and art were principally derived from China. Even the main religious beliefs of the masses of the people have come from China through Chosen. Our dress, our writing, hence our thought have a common origin. Our food is based more upon a vegetarian than a meat diet.

It stands to reason that our physiological standard varies very little. Hence pathological processes ought to run along the same grooves. Medical treatment and incidentally hygienic principles should be based upon the same factors.

And yet, after half a century of progress and intensive research, what do we find? Hippocrates and Aesculapius are still quoted as if they were the only founders of medicine. The name of Shen Nung who laid the foundations of Botany and Pharmacology is practically unknown except among the ultra-conservative. The sound work of the Chou dynasty in Preventive Medicine is not mentioned. No collective and systematic research has been done upon the history of Oriental medicine. True, we have books upon this subject by Fujiwara in Japan and by my friend Dr. Chen Pang-hsien (陳邦賢) in China, but they are not complete and do not represent sufficiently the real contribution towards scientific knowledge by the Orient.

Our anatomy and physiology are still based upon western standards. The teaching of these important subjects with all the faults, even now recognised by thinkers of the West, still goes on. Our peculiarities and variations as met with in the East are not touched upon.

Pharmacology runs along the same western lines, and not enough inquiry has yet been made by our workers. Medicine is approached from an European angle, and the many diseases peculiar to the western world are not properly differentiated from ours. We do not emphasise enough the lesser incidence of gross metabolic diseases as a result of our variations in diet, such as atheroma, aortic disease, angina pectoris, arthritis deformans, gout, rickets, acute rheumatism, etc. In spite of the fact that cancer is often seen in Japan and not rarely in China, I have little doubt that its several forms, particularly of the alimentary tract, occur more frequently in the West.

We still go on treating typhus abdominalis in the same old way, giving milk and barley water to our patients, although they are not used to it. In the treatment of amoebic dysentery, I am sure the West could learn a great deal from the East. The same with many chronic bowel and respiratory diseases and even intestinal parasiticism.



In pediatrics, our students only learn to treat their patients properly long after they have graduated from colleges, because our teachers do not differentiate sufficiently physiological standards in the prenatal, natal and postnatal periods. Tens of centuries of experience have proved that for family and economical reasons children of the East should be weaned after one year, and not six months as western teachers so strongly emphasise.

And now I come to Hygiene, upon the study of which I have devoted the last twenty years. For a long time I have urged for a more liberal study of this great and growing subject. By this, I mean it should be treated as a separate science, not a foster son, so to say, of Pathology or even Bacteriology. I am glad to say this has been done in this University and in Kyoto, where Professors Yokote and Toda have done and are doing such excellent work. In my opinion, Hygiene means more than a mere analysis of milk and water, of ventilation and drainage, of dry complicated statistics. To me, Hygiene is essentially a science, by which and through which all the subjects of medicine could be collectively utilised (1) for the saving of children, and (2) for the prolongation of life. For this purpose, I would emphasise the following points.—

- a.* A radical change in the present method of teaching, making it more practical, more human, and more popular. The masses might then understand and benefit accordingly. Those concerned in the study of communicable disease understand how essential it is for the poor to adopt hygienic methods of living as well as the well-to-do, and how scarlatina, small-pox, cholera, dysentery, typhoid, etc. spare neither rich nor poor, once the virus enters the system.
- b.* The greater use of lectures, pamphlets, ocular demonstrations by means of models, pictures and moving pictures.
- c.* Study of vital statistics, especially epidemiology, from a more accurate standpoint, avoiding such loose terms as deaths from heart failure, blood poison, cough, etc.
- d.* The establishment of popular hygiene museums—big and small—containing easily explained charts and models of food-values, dwellings, economical clothing, disease causation, disease prevention, etc. in every possible aspect.

Emphasise infectious diseases such as cholera, small-pox, phthisis, dysentery; even over-eating, alcohol drinking and uncooked food.

- e. Begin hygienic teaching at home and in the elementary schools.
- f. Take up bravely the question of sexual diseases, and teach their causes and prevention in high schools and colleges.

Particularly are such teachings valuable in China, where the standard of education is still low. The Chinese people are still averse to western treatment, and hence I feel that some other method than mere treatment should be used to benefit them. By their not adopting modern methods of prevention of disease, they are a constant menace to countries which have communication with them. This is of special importance to Japan, which is so near and whose people have so much in common with them. Hence it is to the interest of Japan to lend a helping hand to her big populous neighbor in the promotion of science. China has 400 millions of people but only 10,000 registered doctors. Most modern nations require 1 doctor for every 1,000 inhabitants. Japan has over 60,000 for her 55,000,000 people. So, according to modern requirements, China should have 400,000 doctors.

We have statistics to show that for every 8 persons sick in China, only one comes to the western trained doctor. The other seven either do not see a doctor or go to an old-fashioned man. The one patient visiting a western trained doctor is usually a surgical one (in which case he often gets well if a small operation only is required) but if it were a purely medical case, it would be advanced or chronic, like heart disease or arthritis, and the western man can rarely benefit him.

I am a hygienist and as such believe that the money spent upon small inefficient hospitals and institutions could be better devoted to the training of hygienists, hygienic assistants, lecturers, publication of popular books and pamphlets, and the establishment of museums as outlined above. We may not see immediate results, but I am sure the results will be more permanent, and the new generation will grow up healthier, wiser and better tempered.

In this great work of education, rescue and humanity, Japanese could render immense aid. Our country needs your help and you need our co-operation. This recent earthquake, though sad, has at last enabled us to bury our former distrust



of each other. Chinese are extremely practical and unforgetful. We want to be firm friends with you if only ways and means could be found for mutual benefit.

China is grateful for the decision of your government to use the unexpended portion of the Boxer indemnity of 1901 for the mutual benefit of China and Japan. Leaders of thought like educationists and scientists earnestly hope that this money, amounting still to over two million yen annually, will be devoted to purposes of education and science. For myself, I trust you will allocate at least one half of it for medical purposes, especially, prevention of disease.

Try to win the hearts of the people, rather than a few politicians, and if I may make bold to express my views, they are the following:—

1. Establish more chairs of Chinese language in your centers of learning, so that your people may know and understand the finer aspects of our civilisation and culture. Strange that, although we are so closely related, you have not done this before, for I assure you professorships of Chinese form the nucleus of true friendship and collaboration among the present and future leaders of both nations.
2. Exchange professors between Japan and China. The thinkers and eminent men and women of both countries should see one another more often and cultivate better relations. Send your great men and women now and then over to educational institutions in China and lecture to our teachers and students. In the same way, invite Chinese like Liang Chi Chao, Chu Chi Chien, Wang Ching Chun, etc., to visit this country. All these men have great influence in our respective countries.
3. Establish one or two first class medical colleges with associate hospitals as healthy rivals to the Rockefeller Hospital in Peking. Because the Cantonese have ever been the leaders of great movements in China, I would fix one in the heart of Canton city, so as to show the true friendly feeling of Japan toward them. This is an opportune moment, for there is no first class medical school or hospital yet in Canton.
4. Lastly, I would beg you to establish an Institute of Experimental Medicine, preferably in the Yangtze Valley, say Nanking or Shanghai, where Japanese and Chinese professors could work side by side for

the promotion of research in the East. Most valuable scientific problems still await solution, which I believe Chinese and Japanese could solve better and more economically than western people. Think what a name this will give to Oriental science and what benefit to our respective nations!

5. To this should be added a popular hygiene museum at the center of government, Peking, where our officials and educationists and students may see with their eyes what modern hygiene really means.

The East has still a lot to teach the West in peace, culture, content, and simple life. Let us work together so that even in up-to-date science and research we can accomplish a great deal for the happiness of the world.

WU LIEN-TEH.



**A SURVEY OF PUBLIC HEALTH ACTIVITIES IN CHINA  
SINCE THE REPUBLIC.**

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*Reprinted from National Medical Journal of  
China, March 1923.*

(READ BEFORE C.M.M.A. CONFERENCE, FEB. 15, 1923.)

Modern Public Health activities in China, as in other countries, have largely grown out of emergencies. For instance, the extensive epidemic of Bubonic Plague in Canton and Hongkong in 1894 was mainly instrumental for the promulgation of the Quarantine and other regulations by the Chinese Maritime Customs, which up to now have been the principal authority in China exercising the right of appointing Quarantine and Port Health Officers in the Treaty Ports.

The outbreak of Bubonic Plague in Newchwang in 1898, probably introduced from Hongkong by sea, and a series of cholera epidemics in the Philippines, Japan and Shanghai in succeeding years, helped towards the establishment of Quarantine and Isolation Hospitals at Woosung (Shanghai,) Shanghai City, Taku (Tientsin), Pagoda Anchorage (Foochow), Dairen and Tsingtau.

Another Bubonic Plague epidemic occurring at Tongshan in 1908 was responsible for the establishment of the Peiyang Sanitary Service and the stationing of Medical Officers at Taku, Shanhaikwan and Tientsin by the Chihli Provincial Government.

But it was not until the outbreak of the Great Manchurian Pneumonic Plague, 1910-11, and its aftermath the Mukden International Plague Conference, that the Chinese Government placed Public Health organisation upon a really sound footing by the establishment of the Manchurian Plague Prevention Service and the construction of permanent hospitals at Harbin, Sansing, Lahassusu, Taheiho, Manchouli, Tsitsihar in 1912, and Newchwang in 1919. The Medical Staff of these hospitals undertake the duties of health officers to the local community as well as attending to the general sick. The main laboratory at Harbin also carries on research and manufactures vaccines for plague, cholera, rabies, typhoid, etc.

The Shansi pneumonic plague epidemic of 1918, costing 16,000 lives and invading cities as far south as Pengpu and Nanking, led to the establishment of the Central Epidemic Institute of Peking (located in the grounds of Temple of Heaven). It may be remembered that the Government at that time borrowed one million dollars from the Quadruple Banks for the suppression of the outbreak, used up \$700,000 and devoted the remaining \$300,000 to the founding of this Institute. The organisation was for some years not quite efficient in character being handicapped by insufficient funds and a superabundant staff of non-workers. The appropriation from the Ministry of Interior ran short in 1920, but fortunately the Customs administration came to the rescue and granted an annual sum of \$120,000 for carrying on the work, with the proviso that the Legations of Great Britain, France and Japan appointed their medical officers to serve side by side with Chinese doctors of the Government on the Board of Control. Under this new scheme, matters have improved considerably, and the Institute is now organised so as to include besides the Directors, qualified heads in charge of preventive, biological, chemical and diagnostic departments. Dr. Edgar Tsen of that Institute will speak to you fully on the biologicals prepared by his department.

The Chinese have also established Infectious Disease hospitals managed under modern auspices, as at Peking, Canton, Shanghai and along the different railways, but their number is still far from sufficient. Hand in hand with these developments, one must mention the various activities undertaken by the China Medical Missionary Association and National Medical Association, especially in the foundation of the Joint Council of Public Health Education, which shows a fine output of substantial work to its credit. Most important cities now have their sanitary bureaus, which although their energies are often misdirected under the management of Police Chiefs untrained in Public Health, have made a lasting contribution to the history of hygiene progress in this large country. Nor must we forget the activities of the National Health Association, which, under the greatest difficulties, has rendered much service to Public Health.

Now a few words as to the future outlook of Public Health in this country, especially the share which the C.M.M.A. may profitably contribute towards its enhancement.

It is generally conceded among our missionary as well as non-missionary colleagues that the time has come when a wider view of the demands and possibilities of Public Health should be propagated among all classes of the community. We are



aware that only a small percentage of the sick in China (according to Dr. Balme's statistics only 8 out of every 1000) ever reach the western trained man. The number of physicians during the next few years will still be too small for us to attain a higher percentage. Would it not be worth while for all medical missions to devote part of their time to *preventing* instead of their whole time to *treating* sickness? The immediate results may not be evident, but will nevertheless be more lasting. I would therefore suggest the following intensive campaign:—

*For Missions.* Appoint five to ten percent of their staff for purely public health work, travelling throughout the districts under their influence, make contacts with the local gentry and officials, and work with local Chinese practitioners. Indirectly they will be the means of leading the latter from becoming mere pulse-feelers and drug prescribers, which so many uninfluenced by medical progress have become. Train more nurses, both male and female, for Public Health work.

*For medical schools.* Appoint Professors of Preventive Medicine and place them upon the same footing as those of Medicine and Surgery. Give them equal laboratory and other facilities.

*For associated missions.* Emphasise the need of proper medical inspection in schools and colleges. Advise the authorities to insert health teaching in their curriculum. Wherever possible, emphasise practical details.

*For Chinese institutions.* Volunteer to teach health subjects, examine eyes, compile statistics and even serve as honorary health officers. Cooperate as far as possible with Chinese physicians and authorities.

*For Council of Public Health Education.* Enlarge the scope of the work. Provide ten instead of two active workers by urging the several missions to agree to the seconding of their men and women for this work. Define the districts to be covered by these persons.

*Method to be followed.* A friend of mine especially interested in Public Health tells me that he is in China to sell Public Health. My reply to him is that he must sell it to the Chinese *people*, not to the Chinese *government*. As you know, China is the least governed country in the world, and in spite of revolutions, tuchuns, bandits, and other parasitic offshoots of a materialistic, military, but fortunately temporary phase in the nation's history, her people have remained on the whole calm, dignified and self-supporting. Commerce

has increased in spite of numerous troubles. The Peking and other officials may be corrupt but the masses remain true. Hence, if you want to obtain the maximum of results in Public Health, preach the advantages to the masses, and only use the officials to second, not instigate, your efforts. Also reach the reading classes through books and pamphlets, but for the illiterates you must employ the power of speech and demonstrations. What may be feasible in western countries is often difficult in China and may even have to be discarded. One word more. Our friend, Henry Ford, surveyed America thoroughly before he launched his cheap automobile and made such a success of it. If he or his future prototype were to survey China in the same way, I am sure he would evolve not the *Tin Lizzie* that we know, but what I may venture to call a *motoric*—a machine serving the place of the ricksha but avoiding human labor. Such a motoric should cost under Mex. \$200 to suit all purses in China, have an average fuel consumption of \$15.00 per month, and perhaps not exceed the length of an ordinary ricksha in order to traverse the narrow alleys of Chinese cities.

In the same way, when considering Public Health activities in China I would suggest the following:—

1. Form a Committee of physicians, say five, with experience of Preventive Medicine in China, to devise a program to be approved by the Conference.
2. Request them to emphasise (a) what is practicable in China, and (b) what is attainable within a reasonable period of years.
3. Be specific rather than general. That is, choose for your activities certain centres for experiment. For this purpose may be tried (a) *Canton* where so much has been achieved in spite of adverse circumstances. Take only one aspect of its activities, the utilisation of old burial grounds. As you know, extensive graveyards are usually found studded in the midst of Chinese cities. In the case of Canton, the city fathers, mostly returned students, managed to persuade their fellow-provincials as to the advisability of having the old graves removed to properly laid out places some distance away from the residential quarters. The cost to the Municipality of thus cleaning up one *mou* (one-sixth acre) of ground was \$40, but when the same land (measuring thousands of *mou*) was later put up for auction, each *mou* fetched the magnificent sum of \$5,000. The profits made were used for extending municipal improvements, such as, building



roads, promoting sanitation, cleaning sewers, establishing clinics and erecting up-to-date municipal offices. What other city in the world could boast of such business acumen—all the improvements of the last five years completed without the raising of any municipal loan!

- (b) Harbin, where an efficient medical service has been established for over ten years.
- (c) Nantungchow, the model Chinese city founded by Mr. Chang Chien.
- (d) Tsingtau, recently reverted to China after a proper sanitary service has been maintained by Germans and then Japanese.
- (e) Nanking, where local officials appear unusually goahead.

I have always believed that Christian missions have three duties to perform in this country:—

1. To preach the gospel of Christ.
2. To extend the advantages of science and modern industry so that the masses may not remain so chronically poor.
3. To spread the benefits of medicine, particularly public health.

I may be wrong but feel that the most satisfactory results would be achieved if the order of the above program were reversed, so as to read:—

1. Spread Public Hygiene leading to better health and a clearer mental outlook. This would lead to—
2. Greater physical efficiency, a fuller stomach and keener ambition to succeed. Finally we get—
3. Better appreciation of Christ's words and their truer meaning for China and the world.

WU LIEN-TEH.

## THE STATUS OF MEDICAL EDUCATION IN CHINA IN 1922.

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*Reprinted from China Year Book 1923.*

In order to ascertain the present status of the principal medical schools and colleges in China where a qualification to practise medicine is given, the writer circulated a questionnaire among the sixteen leading institutions, as enumerated in the two accompanying tables. With one exception all replied fully to the questions and these have enabled one to form a fairly accurate idea of present conditions in this country. Briefly summarised, the following information is gleaned:—

1. Oldest college (Naval Med.) started in 1893; newest college (Tsinan) started in 1918.
2. Oldest hospital (Hongkong) established in 1842; newest hospital (Rockefeller, Peking) opened in 1921.
3. Best equipped (Peking Union Med.); least equipped (French-Chinese, Canton).
4. Graduated most students (Army Medical, Peking, 1116 names); graduated least students (Aurora, Shanghai, 4 names).
5. Most expensively-run hospital (Rockefeller, Peking, nearly 1½ million Chinese dollars, according to Director Greene's latest report, 1921).
6. Two hospitals actually showed a profit, namely, Sleeper Davis Hospital for Women, Peking, and the Kung Yee Hospital, Canton.

With a view to studying the actual situation in the several colleges and hospitals, the writer visited almost all individually. Marked difference was noted in the number of buildings, amount and quality of equipment, number of teachers and students, grade of teaching (theoretical and practical), etc. In the prominent ones like the Peking Union Medical, Tsinanfu Medical, the style of buildings and quality of equipment were unrivalled, while in the others, there was practically no laboratory work or hospital (especially in-patient) practice. Following Abraham Flexner's method of classification when dealing with American colleges some twenty years ago, the present writer ventures to classify medical colleges in China in three grades as a result of his inquiries:



*Grade A.* Colleges adequately equipped with buildings, teachers, apparatus, etc., and possessing adequate adjoining hospitals staffed and maintained for proper clinical teaching—

South Manchurian Medical, Mukden,  
Peking Union Medical,  
Christie's Medical, Mukden,  
Hongkong University Medical,  
Hunan-Yale, Changsha,  
St. Johns Univ., Shanghai,  
Christian Univ. Med., Tsinanfu,  
Kung Yee, Canton.

*Grade B.* Either adequate colleges with poor hospital facilities, or adequate hospitals with inadequate colleges—

Army Medical, Peking,  
Naval Medical, Tientsin,  
Special Government Medical, Peking,  
Hackett Women's Medical, Canton,  
Union Medical, Chengtu.

*Grade C.* College and Hospital provisions both inadequate:  
Women's Union Medical, Peking,  
Aurora Medical, Shanghai,  
French-Chinese Medical, Canton.

Besides the above, there exist in China a certain number of first class modern hospitals without any corresponding medical colleges. Of these, the Peking Central Hospital, (founded by Dr. Wu Lien Teh and endowed by Chinese) the Soochow Hospital (established by American Missions) and the Nanking Missionary Hospital deserve honorary mention.

Of mental hospitals (wrongly called lunatic asylums) there is a great shortage, and the only ones available throughout China are:— Canton Missionary, (founded by Dr. Kerr and now managed by Dr. Selden with 500 beds) Peking Municipal (founded and maintained by Chinese with 100 beds) and Shanghai Catholic Infirmary (with 20 beds).

Special Leprosy Hospitals exist in Siao-kan (Dr. Fowler's, near Hankow), Hangchow (Dr. Main), Canton, Foochow, Amoy, etc.

Principal Quarantine Hospitals established by the Chinese Government exist at Woosung (400 beds), Taku (300 beds),

Newchwang (500 beds), Dairen (Japanese, 1,000 beds), Harbin (500 beds), Shanhaikwan (300 beds), etc.

With the rapid growth of modern ideas among the people, the popularity of western medicine has also increased and modern style practitioners may be seen in most cities even far in the interior. But out of about 15,000, only 3,000 are really qualified men and women. Considering that Great Britain with a population of 45,000,000 has at least 50,000 legally qualified practitioners, and Japan with a population of 50,000,000 has over 60,000 licensed practitioners, the number of qualified doctors available for China's needs is exceedingly small. The standard of education must be improved, besides an increase in the number of practitioners.

WU LIEN-TEH.



## A. List of Medical Schools in China.

No.	Name of Medical College	Year founded	Name of founder	No. of teaching staff	No. of students studying	No. of grad. since found.	No. of grad. in 1922 or last grad.	Annual appropriation	Students' fee per annum	Remarks.
1	Army Med. Peking.	1901	Peiyang Govt. (Yuan Shih-kai)	37	340	1113	75	\$115,000	free	
2	Naval Med. T'tsin Special Govt.	1893	Peiyang Govt. (Li Hung-chang)	12 (5E. 7C.)	50	176	24 (1922)	50,000	free	First organized by Dr. Kenneth Mackenzie.
3	Med. Peking, South Manch. Med.	1913	Ministry Educ. Peking.	18	60 ?					
4	South Manch. Med. Mukden.	1911	South Manch. Rly. (Baron Goto.)	29	175	188	31	¥316,200 (1921)	Y3,320	
5	Peking Union Med. Peking	1906	Mission Boards reorganised under Rock. Found. 1919.	56 (Jan. 1922)	(105J. 70C.) 21 under grad.	(131J. 57C.) 105 under previous organization	—	298,515 (1922) M \$394,781	M\$9,000	1st class under new organisation will grad. 1924.
6	Christian Med. Mukden.	1911	D. Christie, C.M.G.	23 (17E. 6C.)	20 grad.	75	28	£5,300 (Ex Eur. sal)	—	Hkong Coll. of Med. founded 1887.
7	Hkong Univ. Med. Dept.	1912	Sir F. Lugard.	18	85	46	11	\$52,132 (Ex. Eur. sal)		
8	Hunan Yale, Changsha.	1914	Hunan Gentry, Yale.	18	45	10	10	G\$50,000, America		
9	St. Johns Univ. Shanghai.	1896	W. H. Boone.	25	34	53	13	M\$50,000, H\$80,000	\$270 ea.	Reduced 1922 to \$175.
10	Christian Univ. Tsinanfu	1918	Mission Boards.	22 (18E. 4C.)	98	29	25 (1921)	\$30,484 (Ex. sal)	\$2,040	
11	Hackett Women's Canton	1899	Mary Fulton.	22 (14E. 8C.)	48	117	6	\$65,000	\$80 ca.	
12	Peking Women's Univ., Peking.	1908	3 Am. Missions.	14	18	47	21	\$4,850	\$50 ea.	
13	Kung Yee, Canton.	1919	Private Society.	29	148	217	12	\$20,000	\$16,000	
14	Union Med. Cheng-tu, Szechuan.	1914	4 Missions.	12	44	7	6 (1922)	\$10,000	\$30 ca.	
15	Aurora, Shanghai.	1911	Society of Jesus.	7 E.	14	4	2	—	\$60 ea.	
16	French-Chinese, Canton.	1905	Paul Doumer.	8 (3E. 5C.)	51	56	8 (1921)	\$12,000	\$3,600	

A.—List of Hospitals in China Affiliated to A.

No.	Name of Hosp.	Year founded	Founder.	No. Med. Staff.	No. Nursing Staff	Out- patients	Annual Outpts.	Annual Expend.	Pts. Receipts.	Remarks.
1	Army Med. Peking.	1918	Minist. War.	20	11	250	7500	\$30,000	Free	No. reply.
2	Naval Med. T'tsin.	1881	Li Hung Chang.	9	4	398 (1921)	55766 (1921)	\$20,000	Mostly free.	
3	Spec. Govt. H. Peking.	1919	Minist. Educ.	10	8	50	—	—	—	
4	South Manch. Ry. Mukden.	1907	South Manch. Ry.	39	92	356	130,209	Y555,427 (1921) Y549,183 (1922)	Y272,069	
5	Peking Union.	1861	London Miss. (Lockhart)	21 (house)	250	140	41,829	£5,000 (Excl. sal.)	\$6,000	
		1915	Rock. Foundation.	24 (visiting)	49 Women (39E. 19C.)	250	71,094	\$360,794	\$110,000 (app.)	
6	Mukden Hosp.	1883	United Free Ch.	18 (16E. 8C.)	23	140	41,829	£5,000 (Excl. sal.)	\$6,000	
7	Hongkong Civil.	1842	Govt.	6	33	150	51,191	?	?	
8	Hunan Yale, Chang-sha.	1906	Yale.	15	14 (6E. 8C.)	450	20,000 (west) 10,900 (east)	\$100,000 (app.)	Mostly free.	
9	St. Lukes, Shanghai.	1866	Archdeacon Thomson.	14	39 (4E. 35C.)	120	9316 (1921)	\$43,279	\$16,890	
10	Christian Univ. Tsinanfu.	1913	Union Miss.	8	24 (4E. 20C.)	153	77,750	\$82,893 (sal. incl.)	\$39,526	
11	David Gregg, Canton.	New 1899	D. Gregg.	(house) 7 (house) 4 (internes)	— 29 (25 Pro)	150 70	37,418	\$50,664	\$21,502	
12	Sleeper Davis, Peking.	Pe-1895	Method. Epis.	5	4	1466	17,097	\$97,717 (Incl. Coll.)	?	
13	Kung Yee, Canton	1911	Chinese Private.	10	34	65	1,285	\$3,000 (Excl. sal.)	\$24,000	profit made by Hospital.
14	Canadian Meth. Chengtu.	1912	Canadian & M. E.	12	3 (E.)	140	17,927	\$73,724	\$80,000	profit made by Hospital.
15	St. Marie.	1906	Catholic.	4	6	105	10,821	\$16,785	\$10,474	
16	Paul Doumer.	1902	P. Doumer.	9 (3E. 6C.)	18	60 100	— 10,135 (1921)	— \$70,000	Free \$15,000	



**RESOLUTIONS PASSED BY THE SCIENTIFIC CONFERENCE HELD IN CHITA, AUGUST 1-3, 1923.**

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*China was represented by Drs. Wu Lien Teh and R. Pollitzer, and Russia by Drs. Barsukoff, Michailoff, Bremm, Pisareff, Sukneff, Jetmar, Kostenko and Krotkoff.*

1. The fight against plague is a matter of great importance, in which all the governments, threatened by plague, are interested. For this reason it is desirable that these governments should work conjointly by holding periodical scientific conferences.
2. The tarabagan is a fundamental epidemiological factor in the spread of plague in Transbaikalia, North Manchuria and Mongolia.
3. One of the most important things in the fight against plague is the detailed investigation—in the plague foci and adjacent districts—of the rodents and their mode of life and also of their parasites: this should be the preliminary condition for carrying out one or the other method for the destruction of the rodents.
4. It is considered desirable to begin in the near future, as a first experiment, the destruction of rodents with poisons and suffocating gases in the plague foci.
5. The prohibition of tarabagan hunting is not considered a practical measure for three reasons: (i) The tarabagan trade has considerable economical importance, (ii) The epizootics are strictly localised to well defined foci and (iii) Negative results are obtained when hunting is prohibited on account of technical difficulties connected with its enforcement.
6. To consider as absolutely necessary the prohibition of hunting in the districts where plague sick tarabagans have been observed.
7. To have thorough medical supervision over tarabagan trade and hunters.

8. To direct the special attention of the persons taking part in the tarabagan trade to dry the skins thoroughly with the aid of sun rays, which latter have a tremendous bactericidal action. The anti-plague organisations will continue to work out a method of thorough disinfection which at the same time does no harm to the skins. Till this matter is settled the skins will be disinfected with formalin gases.
9. The Meeting thinks it useful to direct the attention of the Rockefeller Foundation to the necessity of further investigating the epidemiology of plague, by organisation of expeditions into the plague districts.



**SUMMARY OF TENTH ANNUAL GENERAL REPORT, 1922.**

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Harbin, Oct. 30, 1922.

To His Excellency,  
The Minister for Foreign Affairs,  
Peking.

Sir,—I have the honour to submit a summary of the Tenth Annual General Report of the North Manchurian Plague Prevention Service for the year ending September, 1922.

2.—The end of September of this year marks the completion of the 10th anniversary of our Service. Thanks to the cordial support which we have consistently received from the Central and Provincial authorities, we have been able not only to render considerable services to the sick and suffering in China, but also to place on record before the scientific world a name for Chinese medical research, as evidenced by requests for our Reports and by constant references to our work in medical and scientific literature.

3.—To celebrate the 10th anniversary, we published last September a large medical Report in English and Chinese. The English edition covers 354 pages of reading matter besides a coloured frontispiece of a pneumonic plague lung, three big maps and charts and 36 illustrations. The Chinese edition comprises 198 pages in addition to plates and illustrations. The first half of the book deals with our latest researches connected with the last epidemic of pneumonic plague and includes the following subjects:—

- 1.—The Second Manchurian pneumonic plague epidemic in Manchuria, 1920-21.
- 2.—Observations made during and after the Second Manchurian plague epidemic, 1920-21.
- 3.—The role of the tarabagan in the epidemiology of plague.
- 4.—Clinical observations upon the second Manchurian plague epidemic.
- 5.—Pathological findings in plague pneumonia.
- 6.—Salient points about the 1921 pneumonic plague in Harbin.

Our more important contributions are being published separately in the British Journal of Pathology and Bacteriology, British Journal of Hygiene, China Medical Journal, National Medical Journal of China, American, Japanese and German scientific journals.

4.—About the third week of September, 1922, we received information that some cases of plague had occurred at Haranor in Transbaikalia, 80 versts west of Manchouli Station. Senior Medical Officer J. W. H. Chun together with an Assistant proceeded to the spot on September 23. From inquiries made by Dr. Chun and Dr. Loshiloff, Chief of the Railway Sanitary Department, it was ascertained that a Russian girl, aged 17 years, belonging to a well-to-do family (Tonki,) suddenly took ill early in September with fever and cough and died after nine days. A week later (September 15) the father became sick with shortness of breath and pain in the chest but did not cough up blood. He died on the 17th, followed the next day by his wife whose bloodstained sputum was found by Dr. Krotkoff of Chita to contain plague bacilli. The little daughter succumbed on the 19th and the little son on the 20th, so that within the space of two weeks the whole family of five had died, evidently of pneumonic plague. Specimens sent to our main laboratory in Harbin confirmed the diagnosis. Another case occurring on September 26 at Olovianaya, midway between Manchouli and Chita in Siberia, was also found to be bubonic plague.

One interesting fact gathered from the first series was that the Russian doctor Krotkoff and his assistant were accused by the local populace of having brought the plague and as a punishment were kept inside the infected house for 36 hours (most of the time with the two sick children in one room) before they were released. Fortunately neither prisoner caught the disease and no further cases were recorded in that locality.

The above cases occurring in two different parts of the epidemic regions of Transbaikalia are significant.

5.—Manchuria has been entirely free from plague during the past year. Hongkong, however, claimed an unusual number of bubonic cases (nearly 1,000), though Canton and its environs were practically free. Osaka and Yokohama in Japan also reported a few cases each.

6.—The past year has seen a considerable number of cholera cases in China and Japan. Over 2,000 were recorded in Shanghai district which at one time threatened, as in 1919, to distribute the infection broadcast. Fortunately, thanks to the early precautions taken at Tsingtao, Tientsin, Dairen,



Newchwang and other important centres, the cases, even when they occurred, were limited in number, and a general epidemic was prevented. Less than 20 cases occurred at Tientsin, thanks to the strict measures enforced especially against the sale of melons and uncooked food. In Manchuria, our records were as follows:—

	Cases.	Deaths.
Dairen . . . . .	5 (3J. & 2C.)	2 (1J. & 1C.)
Mukden . . . . .	1 (C)	0 (C)
Newchwang... .	18 (C)	14 (C)
Antung . . . . .	113 (C)	65 (C) up to Oct. 25.

During September a large number of cases were reported throughout Japan and Korea, in spite of the vaccination campaign encouraged by the authorities. The Manchurian port of Antung was infected by cases from the Korean border and has reported to date over 100 cases. It is unfortunate that our proposal to establish a quarantine hospital in that city has not yet matured, so that efficient measures may be undertaken to cope with this epidemic. The establishment of our Quarantine Hospital at Newchwang has probably saved Manchuria from a similar visitation to that of 1919.

7.—Early in January several cases of typhus exanthematicus were reported from Manchouli. I visited Manchouli Station personally to advise our Medical Officer Kwan on the necessary preparations.

Altogether 92 cases were treated between January and April. It is strange that out of a total of 250 cases seen, only three were Chinese, the rest being entirely among Russians.

Influenza invaded Harbin and the Chinese Eastern Railway area about the fourth week of January, apparently as part of the general pandemic. Both Chinese and foreigners were attacked but the disease was on the whole mild, and not many deaths were reported as arising from it. Among other interesting disease seen by us, infectious pneumonia among cattle was prominent. To prevent its spread, inspection and restriction of cattle movement were enforced.

Smallpox and bowel diseases were not unusually frequent this year.

8.—Thanks to the permission given by the Ministry for the allocation of \$30,000 of our reserve funds towards the building of a new patients block, we were able to start the foundations on April 14. Building operations were continued throughout the spring and summer so that the whole block was completed for occupation in the middle of September.

This latest addition to our Hospital is very necessary in view of the gradual tumbling down of the hastily-built structures completed 12 years ago for the great plague epidemic. Besides commodious wards for general 3rd class patients, we have reserved four special rooms for 1st and 2nd class patients. The equipment and sanitary fittings are all up-to-date, in addition to central steam-heating. An X-ray plant is also installed inside the building. A photograph is herewith attached.

The formal opening will take place on October 31 when it is expected that 200 guests will attend.

9.—I am pleased to inform you that 2,400 square sajen (20 mow) of land have been granted to us by the Chinese Eastern Railway on elevated ground in the New Town close to the Russian cemetery. A complete quarantine and observation camp will be erected upon the site as soon as funds are available.

I have also to report that, in reply to our request for a site to build an anti-plague hospital of our own at Manchouli, the railway authorities have granted us a convenient piece of land for the purpose measuring 800 square sajen (5 mow). The premises which we are at present occupying have only been loaned to us by the local Municipal Council, and we trust you will help us in obtaining the necessary appropriation for this vital spot of our anti-plague organization, because plague always invades Manchuria from Siberia through this point.

10.—I obtained three months leave from February to May, which was spent in the Straits Settlements.

The honorary degree of Doctor of Science (Sc. D.) was conferred on June 24 upon me by the St. John's University, Shanghai, "in view of my valuable contributions to medical science and research."

In recognition of the services of our Staff in the suppression of the 1921 plague, the President was pleased to confer various honours upon the members. I received the second class Paokuang Chiaho decoration.

11.—The summer has not been very trying, but the months of August and September were marked by severe typhoons along the coast of China, which did immense damage to all concerned. The towns of Swatow, Taichow, Mokanshan, Chinkiang, Wenchow, Chefoo and Weihaiwei suffered badly. Places as far north as Vladivostok and Kamtchatka were also visited by the typhoons.

12.—The business depression observed all over the East has also affected Harbin. As a result rent has come down



considerably and many lines of goods are unusually cheap. Both railway and municipal authorities have prosecuted their road making programme with vigour, and communications have therefore considerably improved. Our Harbin Hospital, which was formerly rather isolated, can now be reached by good macadamized roads from the New Town, Pristan and Chinese city.

13.—All our station Hospitals report progress. With the exception of Newchwang no cases of cholera were reported at any place. The following are figures of out-patients seen at the respective hospitals in 1921-1922 as compared with previous years:—

	1921-22	1920-21	1919-20	1918-19
Harbin .....	9,345	5,058	11,468	10,474
Taheiho .....	4,290	4,649	6,585	6,513
Sansing .....	4,654	3,631	4,694	3,677
Lahasusu .....	2,213	3,229	1,885	1,089
Newchwang .....	3,043	3,225	3,230	—
Manchouli .....	2,291	—	—	—

The above do not include Customs, Post Office, Government employees and private patients.

Harbin Hospital admitted 208 cases, of which 112 were operations under anaesthetics. Among these were 2 cases of tetanus, 2 of dysentery, 7 of typhoid, 4 of typhus and 8 of influenza.

14.—The following comprise the personnel of the Service during the past year:—

Dr. Wu Lien-teh, Director and Chief Medical Officer.

Mr. R. C. L. d'Anjou, (Commissioner of Customs) Lay Director and Treasurer.

Dr. J. W. H. Chun, M.B., B.C., (Cantab) Senior Medical Officer, Harbin.

Dr. Pollitzer, M.D., (Vienna) Bacteriologist.

Dr. Ando Chu, M.B., CH. B. (Edin.) Senior Medical Officer, Newchwang.

Dr. Liu Chia-swee, in charge of Immunology Dept., Harbin.

Dr. Li Ana, M.D., (Tokio) Parasitologist, Harbin.

Dr. Tang Tsung-nien, M.D., Senior Medical Officer, Taheiho.

Dr. Shih Chi-liang, Resident Medical Officer, Sansing.

Dr. Y. M. Kwan, Resident Medical Officer, Manchouli.

Dr. Chen Ching, Assistant Medical Officer, Harbin.

Dr. Li En-chang, Resident Medical Officer, Lahasusu.

Mrs. Chen Chin-tsai, Deputy Medical Officer, on leave.

Miss Chen Chi-ching, Female Medical Officer, Taheiho.

Miss Kung Sui-chen, Chief Nurse, Harbin.

Miss Chu Tsai-chen, Chief Nurse, Newchwang.

15.—In conclusion, I wish to express my best thanks to the Chinese Government, Chinese Eastern and South Manchurian Railways for facilities granted me in the prosecution of my work. Our relations with the Japanese and Russian Medical Officers have been of a most cordial nature. I am also grateful to our Customs Officers at various stations in Manchuria for frequent assistance rendered to members of our Staff in the course of their duty.

I have the honour, etc.,

WU LIEN-TEH,

*Director & Chief Medical Officer.*



SUMMARY OF ELEVENTH ANNUAL REPORT, 1923.

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Harbin, November 30, 1923.

To His Excellency,  
The Minister for Foreign Affairs,  
Peking.

Sir, I have the honour to submit a summary of the Eleventh Annual General Report of the North Manchurian Plague Prevention Service for the year ending September 1923. Owing to my having had to visit Japan as Exchange Professor during October, this month is for convenience included in the present Report.

2. *Plague.* The past year has been practically free from plague so far as Manchuria is concerned. Three sporadic cases were reported—two in the endemic areas of Transbaikalia and one near Yakoshih, a station on the Chinese Eastern Railway between Mentuho and Hailar. As these cases had a close relation to the epizootics then occurring among the tarabagans (Siberian marmot), a detailed statement may be given here.

Patient 1 was a Russian hunter aged 24. He reported sick on 20th May with high fever and a bubo in right arm-pit. He complained of pain in chest, had slight diarrhoea and cough with blood-stained sputum. He died on 22nd.

Patient 2 was a Russian tarabagan hunter, aged 37 years, and lived 15 versts from Haranor. He had fever on 16th May with a bubo in right axilla, and died on 20th, with characteristic signs of plague.

Patient 3 was a young Russian girl of 12 years living at Station 83 near Dauria (54 versts west of Manchouli). Her family hunted tarabagans and she helped in handling them. She developed a bubo in right axilla and died on 3rd September.

It was significant that all three patients were Russians, all showed buboes in the right arm-pit, and all died of the infection, confirmed later on by animal experiments.

3. After prolonged researches extending for over ten years, we have at last established the connecting link—so to say—between the Siberian marmot and the pneumonic plague outbreaks of 1910-11, 1917-18 and 1920-21. As a result of our investigations conducted this year in Siberia, we are now satisfied that the tarabagan harbours the plague organism in the natural state, which on suitable occasions becomes more virulent and produces epizootics in certain areas. Should human beings encounter such diseased animals, they are liable to contract the disease in a bubonic or septicemic form, which in turn tends to affect the lungs and thus under suitable conditions endangers the community with a pneumonic outbreak.

Working in the endemic regions during the months of May-September of this year, the Chinese-Russian expedition discovered at least eleven tarabagans suffering from plague in the natural state. Such findings have up to now been difficult, because of the short duration of the epidemics among animals, of the rapidity with which eagles and animals of prey devoured sick and dead tarabagans in the fields, and of the wild nature of the country. Other interesting facts established by us during this year were:—

- a. The persistence with which ectoparasites like fleas, lice and ticks, stick to the carcasses and even pelts of the tarabagans.
- b. the possibility of lice as well as fleas conveying plague from animal to animal by bite.
- c. the long duration taken by the plague bacillus to kill the hibernating tarabagan. Three of our animals experimented in winter lived for 73, 41, and 21 days after inoculation. Some showed the disease in a chronic state.

4. *Other Infectious Diseases.* Cholera has been entirely absent from Manchuria and North China this year, though there were small outbreaks in Shanghai and some ports in the south. Scarlet fever invaded many parts of North Manchuria, and severe cases were reported from Taheiho. The type met with was as malignant as that seen in Peking and Tientsin and often killed all the younger members of a family. This disease is worth careful investigation. Dysentery and other bowel diseases were also frequent throughout the summer, and 59 cases of the former were admitted into Harbin Hospital in July. Erysipelas was quite prominent and attacked two members of our medical staff in severe form.



Typhus was practically absent this year. Both Small-pox and Influenza were not unusually common, being quite mild in character.

Cattle plague broke out in a severe form in March-May in the Sui-hua and Lo-pei districts (Heilungkiang). At the request of the Governor we sent two Assistant Medical Officers there, and the epidemic was suppressed by end of June.

5. On 18th August I left Shanghai to attend the fifth Congress of the Far Eastern Association of Tropical Medicine held in this year at Singapore. I officiated as Government delegate from Peking as well as a Vice-President of the Association. Under the presidentship of Dr. A. L. Hoops, Principal Civil Medical Officer, Straits Settlements, the Conference lasted from 1 to 14 September and was attended by representatives from the Philippines, Hongkong, French-Indo-China, Netherlands Indies, Straits Settlements, Malay States, Australia, India, China, Japan, Siam, Formosa, Sarawak, British North Borneo, and Macao. There were 80 oversea members, of whom twelve came from Japan and two from China. The other Chinese delegate besides myself was Dr. Liang Po Chiang of Shanghai.

At this Congress I read two papers, namely,

- a. The Original Home of Plague, and
- b. Plague in Wild Rodents, including latest Investigations upon the Tarabagan.

Much interest was also centered upon the prevention of Beriberi (regarding which the American delegation suggested enactment of laws enforcing the sale of non-polished rice, but this proposal was defeated), control of Malaria, and revised Quarantine Procedure for the Orient. The local British government extended to the visitors every hospitality at Singapore, Kuala Lumpur, Ipoh and Penang, and issued free railway tickets to the official delegates. It was decided to hold the next Congress at Tokio in 1925 with Professor Kitasato as President.

6. I arrived at Shanghai from the south on 7th October, and the same evening left for Nagasaki in order to be in time for the Medical Conference at Kagoshima, to which I had been invited as the principal speaker. My lecture on "Researches upon the Siberian Marmot" lasted one and half hours and was heard by 800 persons, mostly Japanese doctors attending the Conference. After that I visited the Universities of Kyoto, Osaka and Tokio, and delivered a series of addresses upon Hygiene in the Orient, Pneumonic Plague, Inter-depend-

ence of Japanese and Chinese Medicine, etc. Everywhere I was received with the greatest courtesy by government officials, scientists and medical men, and in spite of the recent earthquake was shown every hospitality. This is the first occasion on which a Chinese had been invited to Japan as Exchange Professor, and the opportunity was seized by both sides to cultivate more friendly relations. Whenever possible, I urged my Japanese colleagues to map out new lines of research in medicine and hygiene, which might prove of greater benefit to eastern nations than mere academical and abstract problems. I returned to China on 5th November.

7. Two important meetings were held in the course of the year.

- a. On 14th August, General Chu Ching Ian invited representatives from the Heilungkiang Province, Chinese Eastern Railway and our Service to discuss with him methods for the control of the tarabagan skin trade which had been prohibited by the Tuchun for the past two years. The meeting recommended the establishment of offices at Manchouli and Hailar, where medical inspectors could control the issuance of permits to hunters and the disinfection of skins before export, but the Governor of the Province in a dispatch dated 18th September vetoed this recommendation. Total prohibition of the trade therefore continues, but much smuggling is done across the border, and these skins are now exported via Kalgan and Tientsin.
- b. At the invitation of the medical authorities of Transbaikalia, I left Harbin for Chita on 29th July, and attended a conference initiated by them on 1-4 August. We discussed many aspects of the tarabagan problem, and agreed that, although this animal has now been definitely proved to be the real precursor of plague in those regions, it was essential for economic reasons to regulate, rather than prohibit, hunting and traffic in their skins. Some workable regulations were passed, and I returned on 6th August.

8. I have pleasure in reporting considerable hospital extensions throughout Manchuria. Our Service bought a large very fine stone building at Manchouli for the use of our outpatient clinic as well as quarters from the medical staff. At Newchwang, we have erected a series of permanent brick detention sheds with accommodation for 400 persons. The *kangs* there are of a hygienic nature, the wooden boards resting on



ferro-concrete supports and thus allowing thorough cleaning and disinfection. The total cost is \$35,000.

At Antung, the sum of Tls. 40,000 has been granted by the Customs for the erection of a quarantine hospital at the anchorage spot for steamers on the River Yalu.

At Mukden, a large city hospital—the north-eastern—has been erected by the local authorities at the cost of Mex. \$600,000, consisting of 28 separate blocks and accomodating 400 beds.

At Chihhsien, a fine moderate sized hospital has been built by the Peking-Mukden Railway with Dr. Lumley in charge.

The South Manchurian Railway has commenced operations for the erection of a large railway hospital at Dairen with accomodation for 450 persons. Both architects and contractors are Americans, and the total cost will reach Yen 4,000,000.

In view of these great improvements around us, I earnestly hope that our Service, which has now entered its twelfth year of existence, may be allowed to have more appropriation for its activities. In spite of the increased cost of living and the much enhanced price of all materials, we are receiving the same allowance as twelve years ago.

9. A Commission consisting of Drs. Norman White and Miyajima visited our Harbin Hospital on 27-29 March on behalf of the Health Section of the League of Nations and consulted our staff regarding mutual cooperation in quarantine work and the eradication of epidemics in the Orient. It is hoped that before long the present unsuitable laws enacted in Paris years ago will be changed.

10. The past summer has been unusually wet, floods having on more than one occasion threatened to invade the city of Harbin. In fact the situation looked so bad about the end of August that new dykes were hurriedly built. Fortunately little material damage was done by the rising water. In other parts of Manchuria, miles of railway tracks were washed away, and both the South Manchurian and Peking-Mukden Railways were seriously affected by the floods. The peasants also suffered through damage to their crops. A heavy snowfall occurred at Harbin on 19th October.

11. All our Station Hospitals report progress. No plague or cholera was reported at any spot. The following are figures of out-patients seen at the respective hospitals in 1922-23, as compared with previous years:—

	1922-23	1921-22	1920-21	1919-20	1918-19
Harbin ....	15,343	9,345	5,058	11,468	10,474
Taheiho ...	8,037	4,290	4,649	6,585	6,513
Sansing ...	5,235	4,054	3,631	4,694	3,677
Lahasusu ...	1,470	2,213	3,229	1,885	1,089
Newchwang.	3,693	3,043	3,225	3,230	—
Manchouli...	2,347	2,291	—	—	—

12. The following comprise the personnel of the Service during the past year:—

Dr. Wu Lien Teh, Director and Chief Medical Officer.

Mr. R. C. L. d'Anjou, (Commissioner of Customs) Lay Director and Treasurer.

Dr. J. W. H. Chun, Senior Medical Officer, Harbin.

Dr. R. Pollitzer, Bacteriologist, Harbin.

Dr. Lin Chia Swee, Serologist, Harbin.

Dr. E. B. Young, Senior Medical Officer, Newchwang.

Dr. Tang Tsung Nien, Senior Medical Officer, Taheiho, resigned 30 Sept.

Dr. Shih Chi Liang, Resident Medical Officer, Sansing.

Dr. Y. M. Kwan, Resident Medical Officer, Manchouli.

Dr. Li An, Resident Medical Officer, Manchouli

Dr. Shih Wei Hua, Resident Medical Officer, Taheiho.

Dr. Li En Chang, Deputy Medical Officer, Lahasusu.

Mrs. Y. C. Y. Cheh, Deputy Female Medical Officer, Taheiho.

Miss N. C. Chung, Senior Nurse, Newchwang.

13. In conclusion, I wish to express my best thanks to the Chinese Government, Chinese Eastern and South Manchurian Railways for facilities granted me in the prosecution of my work. Our relations with the Japanese, Russian and Soviet medical officers have been of a most cordial nature. I am also indebted to our Customs Officers at various stations in Manchuria for frequent assistance rendered to members of our staff in the course of their duty.

I have the honour, etc.,

WU LIEN-TEH,  
*Director & Chief Medical Officer.*



## SUMMARY OF THE HARBIN HOSPITAL REPORTS FROM OCTOBER 1921 TO MARCH 1924.

(WITH TWO PHOTOS.)

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1. In March 1922, the Director obtained sanction from the Wai Chiao Pu to build a new block for the housing of patients at a cost of \$30,000. Accordingly the foundations were dug on the 14th April, work was carried on during the whole summer and the building was completed by the middle of October. Altogether there is room for 37 beds (2 first class patients in separate rooms, 5 second class patients, 25 third class, and 5 female patients). The basement consists of a ward, dresser's rooms, boiler room, disinfector room, and animal rooms. The ground floor contains 3 wards, store room, office, operating-room, attendants room and bathroom, while in the first floor there are 4 wards, X-ray room with dark room, two lavatories with one bath, nurse's room, doctor's office and attendants' room.

The whole place is steam heated, well built and fitted with modern sanitary arrangements. A much needed accomodation is thus realized as we have now a hospital that is clean and up-to-date, enabling the staff to take care of the patients in a proper and fitting manner. The X-ray machine is now well housed and can be made to do very useful work.

The large Thresh Disinfector is also placed in an excellent room and is a very tower of strength in the disinfection line.

2. In March 1922, there were many cases of mild influenza both among the Chinese and Russians. The majority of the cases recovered and pneumonia complication was rare.

3. In July 1922, reports of 2000 cases of Cholera were furnished from Shanghai, and 20 cases from Tientsin. Quarantine measures were taken in Dairen and Newchwang where 18 cases were discovered.

In Harbin, we made active preparations against the possible invasion of Cholera. Special blocks were assigned for the housing and treatment of patients and many details in connection with a Cholera campaign were arranged. Luckily no cholera broke out, though many cases of gastro-enteritis, dysentery and typhoid were seen. In one month, we admitted 8 cases of typhoid fever and 7 more were seen among the out-patients. In passing, it is noteworthy that amoebic dysentery is not so rare as some local practitioners assert. They maintain that the bacillary type is far more numerous than the amoebic.

Our laboratory figures are too small to decide this question, but they give one suggestive ideas:—

Amoeba found in dysentery cases 4 in 17 ..... 24%

B. dysentery found in dysentery cases 2 in 17 12%

4. In September 1922, 5 cases of pneumonic plague were reported to have occurred in Haranor, a place 40 versts west of Manchouli, in Soviet territory. We went with Dr. Loshiloff of the Chinese Eastern Railway sanitary staff to investigate. Haranor, a small village near the railway station of the same name, is situated in a well recognized endemic centre. In 1915, there was a small epidemic of plague there. The surrounding country is well stocked with tarabagans among which plague is endemic.

When we arrived there, no more cases were to be seen, as the whole family was wiped out. Effective anti-plague measures were taken by the local authorities and no epidemic resulted.

5. In November 1922, many cases of measles and small-pox were encountered in the city. Two cases of measles with pneumonia complication were admitted into our hospital and they both recovered.

6. Much clinical laboratory work was done by Drs. C. S. Lin and Li An. They do all the necessary tests including the Wassermann reaction for syphilis and were a great help in aiding diagnosis.

7. It was thought that it might be profitable to collect some figures for tuberculosis, syphilis and gonorrhoea cases, composing as they do the majority of our out-patients. The object is to determine what forms of the diseases in question are the most frequent, and what forms are not.

During the 14 months under survey, 2138 cases of tuberculosis, 980 of syphilis and 563 of gonorrhoea were seen as below:—

#### *Tuberculosis*

Lung .....	212	9.9 %
Bones .....	340	15.9 %
Skin .....	384	17.95%
Abdomen .....	4	0.18%
Glands .....	958	45.22%
Testis .....	4	0.18%
Joints .....	152	7.11%
Elsewhere .....	84	3.92%
	<hr/> 2,138 <hr/>	<hr/> 100.1 % <hr/>



*Syphilis.*

1st stage .....	175	17.85%
2nd ,, .....	725	73.97%
3rd ,, .....	80	8.16%
4th ,, .....	0	0. %
	<hr/> 980	<hr/> 99.98%
	<hr/>	<hr/>

*Gonorrhoea.*

Urethra and appendages .....	389	69.09%
Testis .....	53	9.4 %
Joints .....	33	5.86%
Iris .....	6	1.06%
Conjunctiva .....	55	9.77%
Other Organs .....	27	4.79%
	<hr/> 563	<hr/> 99.87%
	<hr/>	<hr/>

8. In February 1923, and again in 1924, many scarlet fever cases appeared. As it is the case elsewhere in China, the type was very virulent, causing many deaths among the patients of whom mostly were children.

Mention may be made of the rarity of diphtheria cases. It is indeed remarkable that so very few are seen. One case was treated with anti-diphtheria serum and recovered in December.

9. A few cases of interest may be cited:—

*a. Compound fracture dislocation of the ankle cured with Bipp.*

A man, 31 years of age, was crushed by a falling bag of beans and felled to the ground. His right ankle was forced inwards, so that there was a compound fracture dislocation of that joint. On admission, there was a wound 3 inches long, through which the articular end of the tibia was thrust. The part was thoroughly cleaned with lysol soap, the ragged edges were cut off. Tinc. Iodine was applied. The tissues were carefully trimmed and coapted. Bipp was smeared all over the wound and the skin was sutured without drainage. The wound healed by first intention and in 6 weeks the limb was quite serviceable.

b. *Haemorrhage into cysternum magnum.*

A man of 40 with a past history of syphilis was taken suddenly ill while stoking a stove and became unconscious.

When he was examined next morning he was comatose, with bilateral exaggerated knee jerks and Babinski sign. As cerebral haemorrhage was suspected, lumbar puncture was performed and almost pure blood was drawn from the spinal canal. Death followed in the same afternoon.

c. *The use of electrargol in Puerperal fever.*

A young married Cantonese woman, 3 para, age 25, was admitted with a history of 4 days fever and rigor starting on the tenth day after an easy and normal parturition.

Her condition pointed to septicaemia and pyaemia. The uterus was large and tender, the lochia was offensive.

Under anaesthesia, the uterus was cleared by blunt curetting and lysol douche. Small pieces of placenta were removed. Immediately after recovering from chloroform, she was seized with violent shivering and became pulseless. Hot water bottles and camphor injection restored her to consciousness and she recovered slowly. Daily injections of 5 c.c. electrargol subcutaneously for 6 days seemed to help to reduce the temperature and the patient was well again on the 13th day.

d. *Carbuncle of the back, one foot square in area.*

A man, 50 years of age, was admitted with the history that a month ago he had a small carbuncle of the back. In spite of treatment by Chinese doctors, it got worse. He had a septic look and was suffering from much pain and the want of sleep, as he could not lie on his back. There was no sugar in the urine. The carbuncle was 1 foot square in area. Under an anaesthetic, the central portion was excised and radiating incisions were made all round. Much pus was evacuated, and as much of the necrotic tissue as possible was removed.

Collosal manganese was injected daily for 7 days. Stannoxyl tablets and large doses of dilute sulphuric acid were given per os and the patient gradually recovered.



e. *A case of liver abscess.*

A soldier, age 27, related that he had severe dysentery 6 months ago, but he was cured in 2 months. One month before admission he started to have fever and pain in the epigastrium with distension of the abdomen. He was thin, and pale but not jaundiced. There was no engorged veins on the abdomen. Some free fluid was detected, the liver was enlarged downwards and upwards; the liver surface was felt to be smooth and hard. The spleen could not be felt. As he had syphilis and also admitted heavy drinking, the question of syphilitic liver or scirrhus had to be considered. His temperature was irregular, and not higher than 101. The blood count showed there were 9000 leucocytes per c.c. However, at the exploratory operation, the abdomen was opened in the right ripple line just below the ribs and at once much ascitic fluid escaped. The liver was large and congested. A puncture showed there was pus. So the trocar was left in the liver and the liver was stitched to the perietal peritoneum, and the abdomen was closed. After draining for two days, the discharge changed from pus to yellow mucus. The trocar was removed and the wound healed by first intention. There was no fever and the pain was no longer felt. Emetine gr. 1 was injected subcutaneously daily for 6 days. The patient became well again at the end of two weeks.

f. *A case of encephalitis lethargica.*

A man, 25 years of age, had slight fever simulating catarrhal fever for 3 days. On the 4th day he felt better and there was no fever. On the 5th day, the temperature, 101°F, returned and this time he had paresis of both legs. Next day he became drowsy and could not be awakened. The left half of the face was paralysed with loss of both knee-jerks. There were no convulsions. The arms were not affected, but the evacuations were not under control. No sugar or acetone was found in the urine. Lumbar puncture showed no pressure, but the fluid was yellowish and opalescent. There were large numbers of white cells. No bacteria could be seen or cultured.

The patient died on the 7th day. This is the only case encountered among the Chinese. There were several reported among the Russians, however.

*g. A case of splenomegaly.*

A young man of 20 came with a history of gradual enlargement of abdomen for 7 years, no history of fever, but with some loss of weight and some pain. On examination he was pigmented in the face with many pimples. His body was not emaciated, but thin. Nothing abnormal was found except that his abdomen was large, the spleen was one inch above the umbilicus, and liver was also felt under the costal margin. His blood and his stool was examined and nothing was found. Arsenic and iron were given with good result after 1½ month's treatment, because he put on weight and the spleen appeared smaller. It was then suggested that the possibility of Kala-azar should be considered, but spleen puncture show no Leishman Donovan bodies, the cultures by Dr. Li were also sterile.

*h. Faecal fistula of the right groin.*

A man of 32 was admitted with a faecal fistula of the right groin. The history was that 3 months ago he had appendicitis with abscess. This abscess burst and a faecal fistula in the groin resulted.

The patient was bed-ridden, thin, yellow and obviously worried. So an operation was performed. A para-medial incision was made and the abdomen was opened. The fistula was found to be at the end of the small intestine near the ilio-caecal valve. An end to end anastomosis was performed and the abdomen closed. Patient made a good recovery and the wound healed well. The site of the fistula is still discharging some serum. All the stool now was passed per rectum.

*i. Two cases of partial occlusion of the vagina.*

Two cases of partial occlusion of the vagina in young women were seen, due to interference of Chinese doctors. The first case was slight and was operated upon successfully. Chinese medicine was inserted into vagina for the treatment of irregular menses with the result that inflammation was set up and the vagina partially closed. The husband refused to have her in the house, so her parent came to get medical help, happily with good result.

The second case was due to prolonged child-birth and manipulation of the midwife. This case was more serious and the result was not so good.





A case of Amyotrophic Lateral Sclerosis.  
Note the wasted muscles and contracture of the flexors.  
症硬變柱旁脊腦性枯肌勢資縮肌及削瘦肉肌示表意注





Case No. III was vesico-vaginal fistula due to prolonged child-birth. There is also some adhesion of the vagina. Two operations have been performed on her with the object of repairing the fistula, but with no success. A third operation also failed.

j. *A case of stab wounds of the chest and abdomen.*

A big strong man said he stabbed himself. Friends of his in the cattle dealer's compound brought him. There were seven knife wounds. The uppermost was in the chest under the heart and wounding the lung, the lowest was in the epigastrium through which a piece of omentum protruded. The others were between these two limits. The wounds were cleaned, iodined, omentum was washed with saline and replaced. The muscles was stitched. All wounds were clipped with Michells clips. They healed by 1st intention and patient left after 2 weeks' stay in hospital.

J. W. H. CHUN,  
*Senior Medical Officer, Harbin.*

## SUMMARY OF FOURTH ANNUAL REPORT FOR NEWCHWANG QUARANTINE HOSPITAL.

Newchwang, June 30th, 1923.

To His Excellency  
The Minister for Foreign Affairs,  
Peking.

Sir,

I have the honour to submit a summary of the Fourth Annual Report of the Newchwang Quarantine Hospital for the year July 1st, 1922—June 20th, 1923.

2. The past year has seen a considerable number of cholera cases in China and Japan. Over 2000 cases were recorded in Shanghai district which at one time threatened, as in 1919, to distribute the infection broadcast. Fortunately, thanks to the early precautions taken at Newchwang, Dairen, Tientsin, Tsingtau and other important ports, the cases, even when they occurred, were limited in number, and a general epidemic was prevented. During September 1922 a large number of cases were reported throughout Japan and Korea, in spite of the vaccination campaign encouraged by the authorities. The Manchurian port of Antung was infected by cases escaping from the Korean border and reported 120 deaths.

3. The port of Newchwang, standing in the direct line of sea traffic from Shanghai, was early menaced, but thanks to proper precautions and the hearty cooperation of the consular body, Customs and civil authorities, we were able to detect and isolate the early cases and thus prevent a repetition of the epidemic of 1919.

Altogether 22 deaths occurred in the whole Newchwang district, eight in our hospital and 14 outside. Of the 21 male and 5 female contacts detained by us, none contracted the disease and all were released well.

A comparison of the cholera cases in the principal ports of Manchuria in 1922 is herewith appended:—

	<i>Cases.</i>	<i>Deaths.</i>
Dairen .....	5 (3J. and 2C.)	2 (1J. and 1C.)
Antung .....	121 (all Chinese)	70 (all Chinese)
Newchwang ...	33 ( „ „ )	22 ( „ „ )
Mukden .....	1 ( „ „ )	0
Harbin .....	Nil	Nil



4. In connection with our anti-cholera measures, the following points may be mentioned:—

Period during which quarantine measures were enforced .....

July 27th to October, 1922.

Number of steamers examined .....	104
Number of passengers examined .....	11,348
Number of junks and sailing boats examined.	1,300
Number of steamers quarantined for suspected cholera ....	one
Number of junks and sailing boats quarantined	one

One corpse found on one steamer proved to be cholera.

One corpse found on the junk proved to be cholera.

Besides the above examinations, we performed a postmortem on a suspected body found on the s.s. *Kansu* on March 27th, 1923. Death in this case was found to be due to strangulated hernia.

5. Of other communicable diseases, a mild type of Influenza prevailed in Newchwang and other parts of Manchuria throughout January and the early part of February, but very few deaths resulted. Small-pox was not abnormally frequent. Plague was entirely absent.

6. From the two enclosed tables of patients treated in our Hospital during the past year, it will be gathered that 2919 out-patients were seen, while 65 were received in the wards, total being 2984. Surgical operations requiring stay in the Hospital numbered eleven.

7. Several improvements and additions have been made during the best year to cope with the growing needs of the Hospital.

These are:—

- a. a mortuary built of red brick, red tiles and cement flooring costing Mex. \$1,200.
- b. a store room with native grey brick and native grey tile roofing and brick flooring, costing Mex. \$276.20.
- c. a large Thresh disinfector purchased from England for £260 has been put in position.

8. Owing to the urgent need of quarters for persons quarantined in time of epidemic, especially during the cholera season, and the unsuitability of the old corrugated iron structures on the north side of the river which were loaned to us two years ago, it was decided by the Commissioner of Customs, Mr. R. L. Warren, and myself to build a series of new permanent buildings for the purpose on our own hospital grounds. Plans were therefore drawn by Mr. Fawcett, Engineer of the Liao Ho Conservancy, and foundations were commenced on June 1st, 1923. The contracted price for the whole construction was small coin \$42,553.00 equivalent to about Mex. \$28,000. There will be four large blocks of brick buildings with cement floors and ferro-concrete supports for my improved *kangs*, each block accomodating 100 persons. It is expected that the building will be ready for occupation on October 31st.

9. Dr. Ando Chu, Senior Medical Officer, resigned in November. Dr. Lin Chia Swee, who was lent from Harbin to Newchwang to take charge of the Bacteriological Department during the cholera out-break, was in charge until December 9th, 1922, when Dr. E. B. Young arrived and took over complete responsibility.

10. I regret to report the death of the Taoyin Ho Hou Chi ( 何厚琦 ) on February 11th, 1923, caused by the effects of charcoal gas poisoning contracted while in Mukden. His funeral took place from the Yamen on March 4th, and was generally regarded as the finest ever held in the city.

The new Taoyin Tung Chao Yuan ( 佟兆元 ) assumed duties on March 11th, having been transferred from the post of Commissioner of Foreign Affairs at Fengtien.

11. The Hospital was honoured by a visit on April 3rd, from Dr. Norman White, Deputy Director, Health Section of the League of Nations, Professor Miyajima (Japanese representative on the Health Section) and Dr. Tsurumi (Head of the Sanitary Department, South Manchurian Railway).

Dr. White was on a tour of investigation of Health Conditions in the Far East with a view to the possibility of applying more uniform quarantine and other health laws by the countries concerned. The visitors were much pleased with the condition and equipment of our hospital for coping with epidemics.

12. Our hospital has been connected with proper water supply from the Japanese water works since October 1922, but owing to some faulty construction and consequent leakage of



the basement in the main building it was not possible to use the central heating. This defect has been remedied during the summer, and it is hoped that all will be well this winter.

13. In conclusion, I wish to acknowledge the cordial support which I have received from R. L. Warren, Commissioner of Customs, whose sound business advice has frequently been invaluable. Dr. Lin Chia Swee, lent by the Manchurian Plague Prevention Service from Harbin, assisted greatly during the cholera invasion. Dr. E. B. Young, Senior Resident Medical Officer and Dr. W. Phillips (Health Officer of the port) have carried out their duties excellently.

I have the honour to be, etc.

WU LIEN-TEH,

*Director and Chief Medical Officer.*

**HARBIN HOSPITAL. 濱江醫院**  
**SUMMARY OF OUT-PATIENTS TREATED.**

From October 1921 to February 1924.

	1921	1922	1923	1924	Total
<i>1. Specific Infectious Diseases 特別傳染病</i>					
<i>(a.) Bacterial Diseases. 細菌病</i>					
1. Typhoid fever 腸室扶斯症	6	41	12	0	59
2. Erysipelas 丹毒	1	7	57	4	69
3. Diphtheria 實扶的里	0	0	8	0	8
4. Pneumonia 肺炎	0	7	36	1	44
5. Influenza 流行性感胃	1	57	141	19	218
6. Whooping cough 百日咳	0	2	30	114	146
7. Gonococcus infections 淋菌傳染病	22	134	428	2	586
8. Dysentery 赤痢症	33	63	168	0	264
9. Cholera 虎列拉	0	0	0	0	0
10. Plague 鼠疫症	0	0	0	0	0
11. Tetanus 破傷風	0	0	3	0	3
12. Leprosy 癩瘋	0	2	0	0	2
13. Tuberculosis 肺結核症	14	213	1,798	182	2,207
<i>(b.) Non-bacterial Fungus Infections. 黴菌傳染症</i>					
<i>(c.) Protozoan Infections. 原生動物傳染症</i>					
1. Malaria 瘧	2	3	42	5	52
2. Relapsing fever 回歸熱症	0	0	0	0	0
3. Syphilis 楊梅毒	125	583	772	156	1,636
4. Yellow fever 黃熱症	0	0	0	0	0
<i>(d.) Metazoan Diseases. 生原虫症</i>					
1. Intestinal Cestodes, Tapeworms					
蠅虫	2	3	30	13	48
2. Diseases caused by Nematodes					
線虫類	2	2	17	0	21
3. Parasitic Insects 寄生虫	2	45	174	23	244
<i>(e.) Infections Diseases of Unknown Etiology. 不知病原傳染病</i>					
1. Small-pox 天然痘	0	0	17	0	17
2. Chicken-pox 水痘	0	1	0	0	1
3. Measles 麻疹	0	4	5	1	10
4. Scarlet fever 猩紅熱	0	0	6	0	6
5. Epidemic Parotitis (Mumps)					
流行性耳下腺炎	1	15	44	6	66
6. Typhus 發疹室扶斯	0	2	0	0	2
7. Rabies 狂犬病	0	2	4	0	6
8. Rheumatic fever 癱麻室扶斯熱症	1	12	0	0	13
9. Acute Tonsillitis 急性扁桃腺炎	2	14	49	5	20
10. Acute Catarrhal fever 急性 加答兒熱症	7	24	88	7	126



	1921	1922	1923	1924	Total
II. <i>Intoxications.</i> 中毒類					
a. Alcoholism 中酒精毒	0	1	0	0	1
b. Morphia Habit 中嗎啡毒	0	13	40	6	59
c. Lead poisoning 中鉛毒	0	7	1	0	8
d. Arsenical poisoning 中砒毒	3	3	3	0	9
e. Food poisoning 中食物毒	0	1	1	0	2
f. Beri-beri 脚氣	0	0	4	0	4
III. <i>Diseases of Metabolism.</i> 新陳代謝病					
a. Gout	0	0	0	0	0
b. Diabetes 糖尿病	0	2	11	1	14
c. Rickets and Scurvy 軟骨及癭症	0	6	0	0	6
d. Rheumatism 癱瘓質斯	25	112	499	115	751
IV. <i>Diseases of the Digestive System</i>					
消化系病	64	576	1,640	117	2,397
V. <i>Diseases of the Respiratory System</i>					
呼吸系病	47	336	827	136	1,346
VI. <i>Diseases of the Genito-Urinary System</i>					
泌尿生殖器病	16	62	321	62	461
VII. <i>Diseases of the Blood</i> 血液病	0	4	128	24	156
VIII. <i>Diseases of the Circulatory System</i>					
循環系病	37	76	99	5	217
IX. <i>Diseases of the Ductless Glands</i>					
無管腺病	0	4	57	22	83
X. <i>Diseases of the Nervous System</i>					
神經系病	36	196	520	104	856
XI. <i>Diseases of the Locomotor System</i>					
運動系病	11	114	160	43	328
XII. <i>Diseases of the Eye</i> 眼科	112	655	845	201	1,813
XIII. <i>Diseases of the Skin</i> 皮膚科	118	551	794	131	1,594
XIV. <i>Diseases of the Nose, Throat &amp; Ear</i>					
耳鼻喉科	38	166	531	178	913
XV. <i>Diseases of Women</i> 婦人科	7	67	123	23	220
XVI. <i>Surgical Cases</i> 外科	858	4,550	5,806	890	12,104
XVII. <i>Vaccination</i> 種痘	5	22	248	0	275
XVIII. <i>Midwifery</i> 產科	0	0	10	1	11
Total.	1,593	8,760	16,597	2,597	29,552

TAHEIHO HOSPITAL. 大黑河醫院  
SUMMARY OF OUT-PATIENTS TREATED.

From October 1921 to February 1924.

	1921	1922	1923	1924	Total
<i>I. Specific Infectious Diseases</i> 特別傳染病					
(a.) <i>Bacterial Diseases.</i> 細菌病					
1. Typhoid fever 腸室扶斯症	0	2	1	0	3
2. Erysipelas 丹毒	0	1	0	0	1
3. Diphtheria 實扶的里	0	0	0	0	0
4. Pneumonia 肺炎	11	8	21	0	40
5. Influenza 流行性感胃	0	1	11	3	15
6. Whooping cough 百日咳	0	0	3	0	3
7. Gonococcus infections 淋菌病傳染	36	43	65	3	147
8. Dysentery 赤痢症	0	4	11	0	15
9. Cholera 虎列拉	0	0	1	0	1
10. Plague 鼠疫症	0	0	0	0	0
11. Tetanus 破傷風	0	0	0	0	0
12. Leprosy 麻瘋	2	0	0	0	2
13. Tuberculosis 肺結核症	0	62	244	11	317
(b.) <i>Non-bacterial Fungus infections.</i> 黴菌傳染病					
(c.) <i>Protozoan Infections.</i> 原生動物傳染症					
1. Malaria 瘧	0	7	8	0	15
2. Relapsing fever 回歸熱症	0	0	0	0	0
3. Syphilis 楊梅毒	52	128	150	11	341
4. Yellow fever 黃熱症	0	0	0	0	0
(d.) <i>Metazoan Diseases.</i> 生原虫症					
1. Intestinal Cestodes, Tapeworms 蠅虫	0	2	0	0	2
2. Diseases caused by Nematodes 線虫類	1	3	4	0	8
3. Parasitic Insects 寄生虫	0	0	10	10	20
(e.) <i>Infectious Disease of Unknown Etiology.</i> 不知病原傳染病					
1. Small-pox 天然痘	0	0	1	0	1
2. Chicken-pox 水痘	0	0	0	0	0
3. Measles 麻疹	0	0	0	0	0
4. Scarlet fever 猩紅熱	0	0	12	1	13
5. Epidemic Parotitis (Mumps) 流行性耳下腺炎	0	0	2	0	2
6. Typhus 發疹室扶斯	0	0	0	0	0



	1921	1922	1923	1924	Total
7. Rabies 狂犬病	0	0	0	0	0
8. Rheumatic fever 僂麻室扶斯熱症	0	0	2	2	4
9. Acute Tonsillitis 急性扁桃腺炎	8	8	7	3	26
10. Acute Catarrhal fever 急性加答兒熱症	2	4	7	0	13
II. Intoxications. 中毒類					
a. Alcoholism 中酒精毒	0	0	4	2	6
b. Morphia Habit 中嗎啡毒	0	0	13	4	17
c. Lead poisoning 中鉛毒	0	0	0	0	0
d. Arsenical poisoning 中砒毒	0	2	1	0	3
e. Food poisoning 中食物毒	0	0	0	0	0
f. Beri-beri 脚氣	0	0	0	0	0
III. Diseases of Metabolism. 新陳代謝病					
a. Gout	0	0	0	0	0
b. Diabetes 糖尿病	0	0	0	0	0
c. Rickets and Scurvy 軟骨及癭症	0	0	0	0	0
d. Rheumatism 僂麻質斯	5	67	56	0	128
IV. Diseases of the Digestive System					
消化系病	38	191	394	20	643
V. Diseases of the Respiratory System					
呼吸系病	17	147	239	16	419
VI. Diseases of the Genito-Urinary System					
泌尿生殖器病	0	54	19	0	73
VII. Diseases of the Blood 血液病	3	4	6	0	13
VIII. Diseases of the Circulatory System					
循環系病	1	10	47	1	59
IX. Diseases of the Ductless Glands					
無管腺病	0	3	2	0	5
X. Diseases of the Nervous System					
神經系病	6	114	124	0	244
XI. Diseases of the Locomotor System					
運動系病	0	12	15	0	23
XII. Diseases of the Eye 眼科	39	324	367	27	757
XIII. Diseases of the Skin 皮膚科	35	169	218	3	425
XIV. Diseases of the Nose, Throat & Ear					
耳鼻喉科	23	49	108	1	186
XV. Diseases of Women 婦人科	5	75	35	1	116
XVI. Surgical Cases 外科	202	1,365	1,506	98	3,171
XVII. Vaccination 種痘	0	66	50	0	116
XVIII. Midwifery 產科	1	0	1	0	2
Total.	492	2,925	3,766	217	7,400

SANSING HOSPITAL. 三 姓 醫 院  
SUMMARY OF OUT-PATIENTS TREATED.

From October 1921 to February 1924.

	1921	1922	1923	1924	Total
<i>I. Specific Infectious Diseases</i> 特別傳染病					
(a.) <i>Bacterial Diseases.</i> 細菌病					
1. Typhoid fever 腸室扶斯症	0	0	0	0	0
2. Erysipelas 丹毒	0	0	4	0	4
3. Diphtheria 實扶的里	0	0	0	0	0
4. Pneumonia 肺炎	0	0	0	0	0
5. Influenza 流行性感胃	2	0	2	0	4
6. Whooping cough 百日咳	0	0	0	6	6
7. Gonococcus infections 淋菌傳染病	10	51	47	0	108
8. Dysentery 赤痢症	2	12	10	0	24
9. Cholera 虎列拉	0	0	0	0	0
10. Plague 鼠疫症	0	0	0	0	0
11. Tetanus 破傷風	0	0	0	0	0
12. Leprosy 麻瘋	0	6	1	0	7
13. Tuberculosis 肺結核症	6	17	21	7	51
(b.) <i>Non-bacterial Fungus infections.</i> 黴菌傳染病					
(c.) <i>Protozoan Infections.</i> 原生動物傳症染					
1. Malaria 瘧	0	0	0	0	0
2. Relapsing fever 回歸熱症	0	0	0	0	0
3. Syphilis 楊梅毒	42	91	78	6	217
4. Yellow fever 黃熱症	0	0	0	0	0
(d.) <i>Metazoan Diseases.</i> 生原虫症					
1. Intestinal Cestodes, Tapeworms 蠅虫	1	3	2	0	6
2. Diseases caused by Nematodes 線虫類	0	0	0	0	0
3. Parasitic Insects 寄生虫	8	29	34	0	71
(e.) <i>Infectious Disease of Unknown Etiology.</i> 不知病原傳染病					
1. Small-pox 天然痘	0	0	0	0	0
2. Chicken-pox 水痘	0	0	0	0	0
3. Measles 麻疹	0	0	0	0	0
4. Scarlet fever 猩紅熱	0	0	0	0	0
5. Epidemic Parotitis (Mumps) 流行性耳下腺炎	0	2	5	0	7
6. Typhus 發疹室扶斯	0	0	0	0	0
7. Rabies 狂犬病	0	0	0	0	0



	1921	1922	1923	1924	Total
8. Rheumatic fever 僂麻室扶斯熱症	0	0	0	0	0
9. Acute Tonsillitis 急性扁桃腺炎	7	36	30	0	73
10. Acute Catarrhal fever 急性加答兒熱症	0	0	0	0	0
II. Intoxications. 中毒類					
a. Alcoholism 中酒精毒	0	0	0	0	0
b. Morphia Habit 中嗎啡毒	2	0	1	0	3
c. Lead poisoning 中鉛毒	0	0	0	0	0
d. Arsenical poisoning 中砒毒	0	0	0	0	0
e. Food poisoning 中食物毒	0	0	0	0	0
f. Beri-beri 脚氣	0	0	0	0	0
III. Diseases of Metabolism. 新陳代謝病					
a. Gout	0	0	0	0	0
b. Diabetes 糖尿病	7	20	21	0	48
c. Rickets and Scurvy 軟骨及癭症	0	0	0	0	0
d. Rheumatism 僂麻質斯	22	157	273	12	464
IV. Diseases of the Digestive System					
消化系病	69	319	571	7	966
V. Diseases of the Respiratory System					
呼吸系病	52	223	224	0	499
VI. Diseases of the Genito-Urinary System					
泌尿生殖器病	0	0	0	0	0
VII. Diseases of the Blood 血液病	0	1	0	0	1
VIII. Diseases of the Circulatory System					
循環系病	8	24	18	0	50
IX. Diseases of the Ductless Glands					
無管腺病	0	0	0	0	0
X. Diseases of the Nervous System					
神經系病	6	10	11	0	27
XI. Diseases of the Locomotor System					
運動系病	4	6	3	0	13
XII. Diseases of the Eye 眼科	54	281	310	14	659
XIII. Diseases of the Skin 皮膚科	125	795	910	240	2,071
XIV. Diseases of the Nose, Throat & Ear					
耳鼻咽喉科	8	43	50	0	106
XV. Diseases of Women 婦人科	16	40	38	0	94
XVI. Surgical Cases 外科	510	2,123	2,657	244	5,534
XVII. Vaccination 種痘	0	0	28	0	28
XVIII. Midwifery 產科	0	0	0	0	0
Total.	961	4,295	5,349	536	11,141

## NEWCHWANG HOSPITAL. 營口醫院

## SUMMARY OF OUT-PATIENTS TREATED.

From January 1923 to February 1924.

	1923	1924	Total
<i>I. Specific Infectious Diseases</i> 特別傳染病			
(a.) <i>Bacterial Diseases.</i> 細菌病			
1. Typhoid fever 腸室扶斯症	0	0	0
2. Erysipelas 丹毒	0	0	0
3. Diphtheria 實扶的里	0	0	0
4. Pneumonia 肺炎	2	0	2
5. Influenza 流行性感胃	0	0	0
6. Whooping cough 百日咳	0	0	0
7. Gonococcus infections 淋菌傳染病	5	0	5
8. Dysentery 赤痢症	2	0	2
9. Cholera 虎列拉	0	0	0
10. Plague 鼠疫	0	0	0
11. Tetanus 破傷風	0	0	0
12. Leprosy 麻瘋	0	0	0
13. Tuberculosis 肺結核症	13	0	13
(b.) <i>Non-bacterial Fungus infections.</i> 黴菌傳染症			
(c.) <i>Protozoan Infections.</i> 原生動物傳染症			
1. Malaria 瘧	4	0	4
2. Relapsing fever 回歸熱症	0	0	0
3. Syphilis 楊梅毒	1	0	1
4. Yellow fever 黃熱症	0	0	0
(d.) <i>Metazoan Diseases.</i> 生原虫症			
1. Intestinal Cestodes, Tapeworms 蠅虫	0	0	0
2. Diseases caused by Nematodes 線虫類	9	0	9
3. Parasitic Insects 寄生虫	86	11	97
(e.) <i>Infectious Disease of Unknown Etiology.</i> 不知病原傳染病			
1. Small-pox 天然痘	1	0	1
2. Chicken-pox 水痘	0	0	0
3. Measles 麻疹	0	0	0
4. Scarlet fever 猩紅熱	0	0	0
5. Epidemic Parotitis (Mumps) 流行性耳下腺炎	1	4	5
6. Typhus 發疹室扶斯	0	0	0
7. Rabies 狂犬病	0	0	0
8. Rheumatic fever 癩麻室扶斯熱症	0	0	0
9. Acute Tonsillitis 急性扁桃腺炎	13	5	18



	1923	1924	Total
10. Acute Catarrhal fever 急性 加答兒熱症	0	0	0
II. Intoxications. 中毒類			
a. Alcoholism 中酒精毒	0	0	0
b. Morphia Habit 中嗎啡毒	2	1	3
c. Lead poisoning 中鉛毒	0	0	0
d. Arsenical poisoning 中砒毒	3	0	3
e. Food poisoning 中食物毒	1	0	1
f. Beri-beri 氣脚	0	0	0
III. Diseases of Metabolism. 新陳代謝病			
a. Gout	1	0	1
b. Diabetes 糖尿病	0	0	0
c. Rickets and Scurvy 軟骨及癭症	0	0	0
d. Rheumatism 僂麻質斯	8	7	15
IV. Diseases of the Digestive System			
消化系病	260	72	332
V. Diseases of the Respiratory System			
呼吸系病	96	8	104
VI. Diseases of the Genito-Urinary System			
泌尿生殖器病	148	11	159
VII. Diseases of the Blood 血液病	1	0	1
VIII. Diseases of the Circulatory System			
循環系病	4	0	4
IX. Diseases of the Ductless Glands			
無管腺病	0	0	0
X. Diseases of the Nervous System			
神經系病	31	7	38
XI. Diseases of the Locomotor System			
運動系病	0	0	0
XII. Diseases of the Eye 眼科	336	27	363
XIII. Diseases of the Skin 皮膚科	220	69	289
XIV. Diseases of the Nose, Throat & Ear			
耳鼻喉科	205	48	253
XV. Diseases of Women 婦人科	18	3	21
XVI. Surgical Cases 外科	1,983	255	2,239
XVII. Vaccination 種痘	36	0	36
XVIII. Midwifery 產科	1	0	1
Total.	3,491	529	4,020

## MANCHOULI HOSPITAL. 滿洲里醫院

## SUMMARY OF OUT-PATIENTS TREATED.

From October 1921 to February 1924.

	1921	1922	1923	1924	Total
<i>I. Specific Infectious Diseases</i> 特別傳染病					
(a.) <i>Bacterial Diseases.</i> 細菌病					
1. Typhoid fever 腸室扶斯症	0	12	2	0	14
2. Erysipelas 丹毒	0	4	0	0	4
3. Diphtheria 實扶的里	0	0	0	1	1
4. Pneumonia 肺炎	0	6	1	0	7
5. Influenza 流行性感胃	0	2	19	5	26
6. Whooping cough 百日咳	0	3	0	0	3
7. Gonococcus infections 淋菌傳染病	0	171	106	27	304
8. Dysentery 赤痢症	0	31	27	0	58
9. Cholera 虎列拉	0	0	0	0	0
10. Plague 鼠疫症	0	0	0	0	0
11. Tetanus 破傷風	0	0	0	0	0
12. Leprosy 麻瘋	0	0	0	0	0
13. Tuberculosis 肺結核症	0	296	155	17	468
(b.) <i>Non-bacterial Fungus infections.</i> 黴菌傳染病					
(c.) <i>Protozoan Infections.</i> 原生動物傳染症					
1. Malaria 瘧	0	1	0	0	1
2. Relapsing fever 回歸熱症	0	0	0	0	0
3. Syphilis 楊梅毒	0	175	484	45	704
4. Yellow fever 黃熱症	0	0	0	0	0
(d.) <i>Metazoan Diseases.</i> 生原虫症					
1. Intestinal Cestodes, Tapeworms 蠧虫	0	11	3	0	14
2. Diseases caused by Nematodes 線虫類	0	0	1	0	1
3. Parasitic Insects 寄生虫	0	0	4	2	6
(e.) <i>Infectious Disease of Unknown Etiology.</i> 不知病原傳染病					
1. Small-pox 天然痘	0	0	0	0	0
2. Chicken-pox 水痘	0	1	0	0	1
3. Measles 麻疹	0	5	0	0	5
4. Scarlet fever 猩紅熱	0	0	0	0	0
5. Epidemic Parotitis (Mumps) 流行性耳下腺炎	0	1	6	0	7
6. Typhus 發疹室扶斯	0	0	0	0	0
7. Rabies 狂犬病	0	0	0	0	0



	1921	1922	1923	1924	Total
8. Rheumatic fever 僂麻室扶斯熱症	0	0	10	3	13
9. Acute Tonsillitis 急性扁桃腺炎	1	3	15	2	21
10. Acute Catarrhal fever 急性加答兒熱症	0	1	1	0	2
II. Intoxications. 中毒類					
a. Alcoholism 中酒精毒	0	1	0	4	5
b. Morphia Habit 中嗎啡毒	1	2	1	0	4
c. Lead poisoning 中鉛毒	0	0	0	0	0
d. Arsenical poisoning 中砒毒	0	0	0	0	0
e. Food poisoning 中食物毒	0	1	0	0	1
f. Beri-beri 脚氣	0	0	0	0	0
III. Diseases of Metabolism. 新陳代謝病					
a. Gout	0	1	0	0	1
b. Diabetes 糖尿病	0	0	0	0	0
c. Rickets and Scurvy 軟骨及癰症	0	0	0	0	0
d. Rheumatism 僂麻質斯	0	42	79	19	140
IV. Diseases of the Digestive System. 消化系病	5	257	339	55	656
V. Diseases of the Respiratory System. 呼吸系病	6	93	155	51	305
VI. Diseases of the Genito-Urinary System. 泌尿生殖器病	0	10	18	0	28
VII. Diseases of the Blood. 血液病	0	2	1	0	3
VIII. Diseases of the Circulatory System. 循環系病	0	2	30	9	41
IX. Diseases of the Ductless Glands. 無管腺病	4	12	0	0	16
X. Diseases of the Nervous System. 神經系病	0	41	69	9	119
XI. Diseases of the Locomotor System. 運動系病	0	0	7	1	8
XII. Diseases of the Eye. 眼科	6	274	283	29	592
XIII. Diseases of the Skin. 皮膚科	10	296	228	20	554
XIV. Diseases of the Nose, Throat & Ear. 耳鼻喉科	0	30	59	12	101
XV. Diseases of Women. 婦人科	0	19	21	14	54
XVI. Surgical Cases. 外科	0	359	326	140	815
XVII. Vaccination. 種痘	0	12	0	0	12
XVIII. Midwifery. 產科	0	0	0	0	0
Total.	33	2,177	2,440	465	5,115

## LAHASUSU HOSPITAL. 拉哈蘇蘇醫院

## SUMMARY OF OUT-PATIENTS TREATED.

From October 1921 to February 1924.

	1921	1922	1923	1924	Total
<i>I. Specific Infectious Diseases 特別傳染病</i>					
<i>(a.) Bacterial Diseases. 細菌病</i>					
1. Typhoid fever 腸室扶斯症	0	0	0	0	0
2. Erysipelas 丹毒	0	0	3	0	3
3. Diphtheria 實扶的里	0	0	0	0	0
4. Pneumonia 肺炎	0	0	0	0	0
5. Influenza 流行性感冒	0	0	4	3	7
6. Whooping cough 百日咳	0	6	27	2	35
7. Gonococcus infections 淋菌傳染病	11	13	0	0	24
8. Dysentery 赤痢症	7	68	36	0	111
9. Cholera 虎列拉	3	0	0	0	3
10. Plague 鼠疫症	0	0	0	0	0
11. Tetanus 破傷風	0	0	0	0	0
12. Leprosy 痲瘋	0	4	0	0	4
13. Tuberculosis 肺結核症	8	7	16	1	32
<i>(b.) Non-bacterial Fungus infections. 黴菌傳染病</i>					
<i>(c.) Protozoan Infections. 原生動物傳染症</i>					
1. Malaria 瘧	0	0	5	0	5
2. Relapsing fever 回歸熱症	0	0	0	0	0
3. Syphilis 楊毒梅	42	90	19	14	165
4. Yellow fever 黃熱症	0	0	0	0	0
<i>(d.) Metazoan Diseases. 生原虫症</i>					
1. Intestinal Cestodes, Tapeworms 蠧虫	0	41	0	0	41
2. Diseases caused by Nematodes 線虫類	6	0	0	0	6
3. Parasitic Insects 寄生虫	0	0	0	0	0
<i>(e.) Infectious Disease of Unknown Etiology. 不知病原傳染病</i>					
1. Small-pox 天然痘	0	4	0	0	4
2. Chicken-pox 水痘	0	0	0	0	0
3. Measles 麻疹	0	20	6	0	26
4. Scarlet fever 猩紅熱	0	0	0	0	0
5. Epidemic Parotitis (Mumps) 流行性耳下腺炎	0	0	0	0	0
6. Typhus 發疹室扶斯	0	0	0	0	0
7. Rabies 狂犬病	0	0	0	0	0
8. Rheumatic fever 僂麻室扶斯熱症	0	0	0	0	0



	1921	1922	1923	1924	Total
9. Acute Tonsillitis 急性扁桃腺炎	10	11	15	0	35
10. Acute Catarrhal fever 急性加答兒熱症	0	0	0	0	0
II. Intoxications. 中毒類					
a. Alcoholism 中酒精毒	0	0	0	0	0
b. Morphia Habit 中嗎啡毒	2	0	0	0	2
c. Lead poisoning 中鉛毒	0	0	0	0	0
d. Arsenical poisoning 中砒毒	1	0	0	0	1
e. Food poisoning 中食物毒	0	0	0	0	0
f. Beri-beri 腳氣	0	0	3	0	3
III. Diseases of Metabolism. 新陳代謝病					
a. Gout	1	5	0	0	7
b. Diabetes 糖尿病	2	0	0	0	2
c. Rickets and Scurvy 軟骨及癭症	3	5	0	0	9
d. Rheumatism 僂麻質斯	7	16	40	0	63
IV. Diseases of the Digestive System					
消化系症	50	137	151	9	347
V. Diseases of the Respiratory System					
呼吸系病	16	0	0	0	16
VI. Diseases of the Genito-Urinary System					
病泌尿生殖器	0	0	0	0	0
VII. Diseases of the Blood 血液病	8	10	27	0	45
VIII. Diseases of the Circulatory System					
循環系病	0	0	0	0	0
IX. Diseases of the Ductless Glands					
無管腺病	0	0	0	0	0
X. Diseases of the Nervous System					
神經系病	0	0	1	0	1
XI. Diseases of the Locomotor System					
運動系病	0	0	0	0	0
XII. Diseases of the Eye 眼科	30	115	106	6	257
XIII. Diseases of the Skin 皮膚科	22	112	144	4	282
XIV. Diseases of the Nose, Throat & Ear					
耳鼻喉科	16	16	31	6	69
XV. Diseases of Women 婦人科	13	101	84	5	203
XVI. Surgical Cases 外科	118	642	594	58	1,412
XVII. Vaccination 種痘	0	56	55	0	111
XVIII. Midwifery 產科	0	0	0	0	0
Total.	376	1,481	1,367	108	3,332

SUMMARY OF THE IN-PATIENT DISEASES.

From October 1921 to February 1924.

住院患者各症之分類表自一九二一十月至一九二四二月底止

	Harbin Hosp.	Takeiho Hosp.	Newchwang Hosp.
	哈爾濱醫院	大黑河醫院	營口醫院
Fracture and Dislocations. 骨折及節脫			
Fr. Clavicle 鎖骨	1	—	—
„ Humerus 上膊骨	4	—	—
„ Wrist 腕	2	—	—
„ Thigh 大腿	6	—	—
„ Tib. & Fib. 脛及腓骨	13	3	2
„ Ankle 踝節	1	—	—
Dislocation hip 關節脫臼	1	—	—
Fr. Spine 脊柱骨折	1	—	—
„ Elbow 肘	1	—	—
„ Skull 顱骨	1	—	—
„ Rib 肋骨折	1	—	—
Dislocation shoulder	3	—	—
Injuries. 外傷			
Sprain 扭傷	3	—	—
Contusions 挫傷	18	5	5
Frost bite 凍傷	3	—	—
Kick 馬踢	2	—	1
Bites 咬傷	2	—	—
Gun shot 槍彈傷	42	10	1
Bomb explosious 炸彈傷	1	—	—
Crushes 壓傷	1	—	—
Cut throat 頸處自刎傷	2	—	—
Concussion 受震	1	—	—
Stabs & wounds 刺及槍	34	4	4
Scalds 燙傷	2	—	—
Burn 火傷	6	—	—
Diseases of Genito Urinary. 生殖及泌尿器病			
Nephritis 腎炎	6	—	—
Phimosis 包莖	4	—	—
Stricture urethra 尿管狹窄	2	—	—
Bubo 橫痃	9	—	—
Gonorrhoea 淋病	4	—	5
Incontinence urine 遺尿症	1	—	—
Hydrocele 陰囊水腫	1	—	—



	Harbin Hosp.	Takeiho Hosp.	Newchwang Hosp.
	哈爾濱醫院	大黑河醫院	營口醫院
Orchitis 睪丸炎	3	—	3
Stone urethrae 尿道石	2	—	—
Syphilitic chancre 下疳	3	—	—
Soft chancre 軟下疳	1	—	—
Cystitis 膀胱炎	1	—	—

*Diseases of Alimentary Canal and Digestive System.* 消化管及消化系病

Colitis 腸絞	—	2	—
Dysentery 赤痢	3	—	—
Typhoid 腸室扶斯症	11	2	2
Enteritis 腸炎	—	1	—
Cirrhosis liver 肝變硬症	2	—	—
T. B. Peritonitis 腹膜結核	1	—	—
Hernia 疝氣	5	—	—
Piles 痔核	40	2	3
Fistula in ano 痔漏	51	2	4
Fissure anus 肛門裂	1	—	—
Constipation 便秘	1	—	—
Liver abscess 肝瘡	1	—	—
Gastritis 胃炎	3	—	—
Dyspepsia 消化不良	7	—	2
Appendicitis 闌尾炎	3	2	—
Gastroptosis 胃下垂	2	—	—

*Diseases of Women.* 婦人病

Puerperal septicaemia 產褥熱	1	—	—
Prolapse uterus 子宮墜	1	—	—
Gonorrhoea 淋病	7	—	3
Endometritis 子宮內膜炎	2	—	1
Occlusion of vagina 陰道閉塞	2	—	—
Vesico-vaginal fistula 陰膀胱漏	2	—	—

*Diseases of Circulatory and Respiratory System.* 循環及呼吸系病

Pneumonia 肺炎	7	2	—
Empyema 膿胸	3	—	—
Mitral 僧帽瓣症	4	—	1
Phthisis 肺結核	18	3	1
Pleurisy 肺膜炎	3	—	—
Bronchitis 氣管枝炎	5	—	—
Aortic disease 總脈弓症	—	—	1
Pharyngitis 喉炎	2	—	—
Popliteal aneurysm 腘脈囊	3	—	—

		Harbin Hosp.	Taheiho Hosp.	Newchwang Hosp.
		哈爾濱醫院	大黑河醫院	營口醫院
Asthma	哮喘	5	—	—
Varicose veins	靜脈曲張	—	2	—
Emphysema	肺氣腫	1	—	—
Endocarditis	心囊炎	1	—	—
<i>Tumours.</i> 瘤				
Granuloma	肉瘤	2	—	—
Fibroma	纖維瘤	8	—	—
Sarcoma	肉腫	7	—	—
Carcinoma	癌腫	3	—	—
Papilloma	荊瘤	1	—	—
Epithelioma	上皮癌腫	2	—	—
Cyst	囊腫	5	2	—
Keloid	癭瘤	—	—	1
Condyloma	楊梅濕粒	2	—	—
Lipoma	脂肪瘤	1	—	—
Cystadenoma	囊狀腺腫	2	—	—
Ranula	舌下軟瘤	1	—	—
<i>Diseases of Nervous System.</i> 神經系病				
Peripheral neuritis	週圍神經炎	—	2	—
Myelitis	脊髓炎	2	—	—
Amyotrophic lat. sclerosis	硬變性偏癱症	1	—	—
Spastic paraplegia	肌枯性脊髓變硬症	1	—	—
Encephalitis lethargica	腦腦炎	1	—	—
Cerebral meningitis	腦腦衣炎	2	—	—
Insomnia	失眠	2	—	—
Epilepsy	癇	2	—	—
Hysteria	癡	2	1	2
Cerebral Haemorrhage	大腦流血	4	—	1
<i>Septic Cases.</i> 化膿症				
Abscesses	膿瘍	42	2	5
Ulcers	潰瘍	10	1	1
Gangrene	壞疽	1	—	—
Eczema	濕疹	2	1	—
Tetanus	破傷風	1	—	1
Lupus	狼瘡	3	—	—
Carbuncle	癰	7	—	—



	<i>Harbin</i> <i>Hosp.</i> 哈爾濱醫院	<i>Taheiho</i> <i>Hosp.</i> 大黑河醫院	<i>Newchwang</i> <i>Hosp.</i> 營口醫院
Erysipelas 丹毒	3	—	—
Furunculosis 癰病	3	—	1
Gumma 楊梅瘤	1	—	—
Dermatitis 皮炎	1	—	—
Scabies 疥	2	—	—
Cellulitis 連翹炎	12	—	4

*Diseases of the Eye.* 眼病

Corneal ulcer 瞭瘍	3	—	—
Pterygium 胬翳	1	—	—
Pannus 絡瞭	1	—	1
Entropion 臉捲內	2	—	—
Trachoma 胬粒炎	16	—	—
Gon. ophthalmia 淋毒眼	3	—	—
Ectropion 臉捲外	1	—	—
Keratitis 瞭炎	1	—	—
Retinitis Pigmentosa 視衣黑點症	1	—	—
Conjunctivitis 胬炎	2	—	3
Cataract 障	4	—	1

*Diseases of Bones, Muscles and Joints.* 骨筋及關節病

T. B. Bone 骨結核	45	4	8
T. B. Joint 關節結核	12	—	—
Necrosis 骨死	8	1	—
Periostitis 骨膜炎	1	—	1
Suppurative arthritis 關節化膿炎	2	—	—
Gon. arthritis 淋症節炎	1	—	—

*Fevers.* 熱病

Typhus 發疹室扶斯	5	—	—
Influenza 流行性感胃	14	—	—
Malaria 瘧	1	—	—
Catarrhal 加答兒炎			
Scarlet fever 猩紅熱	2	—	—
Tonsillitis 扁桃腺炎	3	—	—
Measles 麻疹	2	—	—
Acute rheumatism 急性僂瘋室斯	2	—	—
Diphtheria 實扶的里	1	—	1

	<i>Harbin</i>	<i>Taheiho</i>	<i>Newchuang</i>
	<i>Hosp.</i>	<i>Hosp.</i>	<i>Hosp.</i>
	哈爾濱醫院大	黑河醫院	營口醫院
<i>Various.</i> 雜症			
Rheumatism 癱麻質斯	7	—	1
Starvation 凍死	2	—	—
Pregnancy 孕	18	2	4
Carbolic poison 煤酸毒	1	—	—
Corns 釘疥	1	—	—
T. B. Glands 腺結核	18	—	3
Occlusion nostrils 鼻孔閉塞	2	—	—
Splenomegaly 脾大	3	—	—
Coal gas poison 煤氣毒	2	—	—
2nd. Syphilis 第二期梅毒	10	—	1
Anaemia 血虧	2	—	—
Hare lip 兔唇	1	—	—
Supernumerary digit 六指	1	—	—
Opium habit 中鴉片癮	1	—	—
Tetanus 瘧	1	—	—
Noma 潰瘍	1	—	—
Diabetes 尿淋症	1	—	—
Mercury poison 中錄毒	2	—	—
Opium poison 中鴉片毒	—	—	1
No diagnosis 未疹斷	5	—	5
	—	—	—
	738	56	85
	—	—	—



LIST OF OPERATIONS BETWEEN OCTOBER 1921 TO  
FEBRUARY 1924, HARBIN HOSPITAL.

哈爾濱醫院手術表一九二一十月至一九二四年二月底止

Amputations.	肢截斷術		Carbuncle	癰	1
			Bullet extraction	取彈	13
Toe	脚趾	6	Cellulitis	連翹炎	2
Fingers	手指	14	Tenotomy	腱割斷術	1
Arm	臂	9	Lupus	瘰	1
Foot	足	3	Tumours.	瘤	
Leg	小腿	20			
Thigh	大腿	9	Fibroma	纖維腫	11
Hand	手	1	Cyst	囊腫	7
Penis	陽莖	3	Sarcoma	肉腫	2
Breast	乳房	1	Papilloma	癌腫	5
Bones and Joints.	骨及關節		Granuloma	肉芽瘤	5
			Ovarian cyst	卵腺癰瘤	1
Necrosis	死骨	12	Bronchial cyst	氣管支癰	1
Compound fractures	複雜骨折	6	Haematoma	血瘤	3
Scraping T. B. Bone	刮骨結核	18	Keloid	癭瘤	1
Plating fractures	釘接骨股	1	Sebaceous cyst	脂癰	1
Resection rib	割取肋	2	Alimentary Canal and Abdomen.		
Sequestia of skull	割取髑死骨	2			
Genito-Urinary.	生殖器及尿道			育道及腹	
			Piles	痔核	27
Circumcision	環狀切包莖	6	Fistula in ano	痔漏	54
Castration	割去睪丸	5	Wound of abdomen	腹傷	1
Hydrocele	腎囊	1	Hernia	疝氣	4
Urethral Stone	尿道石	2	Exploratory laparotomy	剖腹	5
Stricture urethrae	尿道狹窄	3	Extraction of tooth	拔牙	3
Stenosis vagina	陰門狹窄	2	Fissure anus	肛門裂	2
Blood Vessels.	血管系		Tonsils and adenoids	扁桃腺	2
			Intestinal anastomosis	腸相通法	1
Ligature Femoral artery	縛腿脈	1	Peritonitis, drainage	腹膜炎 引流	1
Skin, Fascia and Tendons.			Needle in abdomen	針刺入腹	1
			Liver abscess	肝癰	1
	皮筋鞘肌腱		Eye.	眼	
T. B. Glands	腺結核	14			
Cleaning wounds	潔淨傷	2	Ectropion	眼外捲	3
Perineal fistula	會陰漏	1	Entropion	眼內捲	3
Abscess	膿瘍	21	Cataract	障	1
Needle in hand	針入手中	4	Excision eye	眼剜割術	5
Suture wounds	縫合傷	6	Pterygium	胛翳	1

# 314 MANCHURIAN PLAGUE PREVENTION

## Plastic. 成形術

Excision ulcer	割取瘍	1
Double hare lip	兔唇	1
Occlusion nostrils	鼻孔塞	1
Ingrowing nail	甲邊入肉	1

## Various. 雜類

Examination under anaesthetic	施麻醉疹查	5
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Forceps delivery 施產鉗收生 2

Intra-thecl medication 脊鞘內 1

Intra-ulterine douche 洗子宮 1

Extraction Indian corn from ear

取耳內玉蜀米 1

Manipulation under anaesthetic

痲醉後施手術 2

Total 總數 361



# 圖点中疫鼠生發亞細亞北東

Map of North Eastern Asia showing important Plague Centres.

